Again and again through the history of civilization whole countries, continents even have been swept by rapidly spreading bacterial or virus borne diseases some deadly, some crippling some less destructive The Black Death of the Middle Ages the Great Plague of 1665 the cholera epidemics of the nineteenth century the more modern scourges of influenza in its various forms and poliomyelitis are but a few of the best known

This book tells the story of the ravages caused by these diseases in the past of the origin spread and effects of epidemics, and the attempts more or less successful to combat them It discusses immunity and immunization the steps taken in recent times through the growth of public health services and general medical knowledge to prevent and forestall the recurrence of such scourges such as leprosy smallpox and tuberculosis and the possibility of their return in the future

PELICAN BOOKS

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EPIDEMIC DISEASES

A H GALE



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FOREWORD

THIS book on pestilence through the ages was almost completed when Arthur Gale died so prematurely in 1956 I was asked by Mrs Gale to edit the volume a task which I gladly accepted and which has given me great personal pleasure

The work has not proved easy as although conversant with much of the book since its conception I had little knowledge of the sources to which Arthur Gale had turned to compile this fund of knowledge.

I should like to express my thanks to Professor Bruce Perry for much helpful advice and for writing the preface to this work Also to John Lane The Bodley Head Ltd for permission to include certain plates and diagrams contained in a book by Walter G Bell F.S.A. F R A S entitled *The Great Plague of London in 1665* and published by The Bodley Head

E R HARGREAVES

PREFACE

When Dr Arthur Gale died in October 1956 he left the nearly completed manuscript of this book which represents an expression of the main guiding interest of his professional life The changes in the epidemiology of the common infectious diseases had interested him for many years Always keenly interested in the welfare of children he was from 1933 to 1947 one of the medical officers to the Board of Education Some of his researches during this time formed the basis of his Milroy Lectures to the Royal College of Physicians in 1944 on A Century of Changes in the Mortality and Incidence of the Principal Infections of Childhood His increasing interest in epidemiology lead in 1947 to his transfer to the Ministry of Health There he became a member of the epidemiological team and was thus enabled to continue his studies on the epidemiological features of acute anterior poliomyelitis In 1950 he was invited by the University of Bristol to accept the combined posts of Director of Medical Post Graduate Studies and Lecturer in Epidemiology The former post brought him into contact with consultants and practitioners throughout the West Country and this enabled him with their help to continue his researches He was particularly intrigued by the way in which epidemics of anterior poliomyelitis widely separated in time struck the same villages in the south west, and he spent much time and thought in attempting to solve this problem. This book written in simple and non technical terms introduces to the layman the fascinating history of the common infectious diseases in this country After Dr Gale s death the manuscript has been completed and in a few places brought up to date by one of his great friends Dr E R Hargreaves Deputy Medical Officer for the County of Cornwall It is largely due to Dr Hargreaves s enthusiasm and devoted care in preparing the manuscript that the book is now published It forms a most fitting memorial to Dr Gale s life work.

C. Bruce Perry m d f r c p

INTRODUCTION

There are many books on the history of medicine and several on the history of public health but the only book which deals systematically with the history of epidemic diseases in this country is Creighton s great work of scholarship *The History of Epidemics in Britain* published in two volumes in 1894 Creighton s book is an inexhaustible store of information but it is not the kind of book which anyone would read unless he had a special interest in the subject Greenwood s *Epidemics and Crowd Diseases* is the nearest approach to a history for the general reader but the first part of it is concerned with the development of epidemiological theory from the time of Hippocrates and the second treats of individual diseases as examples to illustrate epidemiological principles Thus Greenwood s book is in some ways more comprehensive than this one and in others less He ranges the world for his examples but he does not include all the epidemic diseases which have at some time been important in Britain

In an address to the National Book League Trevelyan once said Besides the contemplation and study of the past for its own sake there remains the second great value of history namely the light it throws on the present There can be little doubt about the intrinsic interest of the history of epidemic diseases but there may be some about the value of this study for the light it throws upon the present because many of the diseases to be discussed have vanished from the English scene or have become trivial However infantile paralysis is still with us we can never be quite sure what influenza may do and several of the other diseases cause a great deal of sickness even if they no longer kill as they used to do

The chief criterion for the inclusion of a disease has been that it has at some time been epidemic in the dictionary sense of being prevalent at one time and not at another Some diseases which are certainly epidemic for example

epidemic process is fundamentally the same as that which the gardener sees when his strawberry plants or his raspberry canes proceed to die off from a virus infection

Animals including man have a highly complex mechanism for protecting themselves against attack by micro-organisms and producing immunity and the following scheme gives some idea of how this works

- Immunity
  - Innate
  - Acquired
    - Passive
    - Active

Not very much is known about innate immunity but it obviously exists because some micro-organisms have power to produce disease in many different kinds of animals whereas others affect only one kind It seems possible that there may be genetic differences between individual human beings which determine to some extent their susceptibility to different diseases but we have little exact knowledge about this

Acquired immunity is dependent on the fact that the introduction of the parasite into the host results in the production of protective substances The substances in the parasite which provoke this response are known as antigens and the substances produced by the host are called antibodies The details of antigen-antibody reactions in special cases are extremely complicated, and a large proportion of the time of bacteriologists is spent in working out such details but for our purpose it is sufficient to know that antigen provokes the production of antibody that a single micro-organism may incorporate a number of different antigens and that variations in the antigenic structure of some micro-organisms frequently occur both under natural conditions and in the laboratory

We speak of immunity as being active when the host has itself manufactured antibodies as a result of invasion by a parasite and we speak of it as passive when some other host has done the manufacturing and the ready made antibodies are transferred from this host to the one we wish to protect Active immunity may occur naturally as a result of infection or be produced artificially by the administration of some

chicken pox and mumps, have perforce been omitted because they have led such blameless lives from the point of view of causing serious damage that they have little recorded history

All the diseases to be discussed are believed to be due to micro organisms and although my aim has been to write a book which shall be intelligible to the general reader, it is hardly possible to avoid the use of some technical terms in writing about them

Each chapter begins with a short summary of modern knowledge about the disease or diseases to be discussed but the following general remarks may help the non technical reader to understand these summaries Most of the micro-organisms which cause important epidemic diseases in human beings in Britain belong to two main classes (1) the bacteria or (2) the viruses The bacteria belong to a world in which the unit of measurement is the micron i e one thousandth of a millimetre – written 1 μ They are visible directly under the highest power of an ordinary microscope The viruses belong to a world in which the unit of measurement is only one thousandth of 1 μ – written 1 mμ – but they vary in size more than the bacteria the largest of them being about 300 mμ in length or diameter whereas the smallest are about 10–20 mμ The largest viruses may be just visible under the highest power of an ordinary microscope but most of them can be made visible only in photographs taken with an electron microscope Bacteria can multiply outside the body and are habitually grown in enormous numbers in or on various substances in thousands of bacteriological laboratories throughout the world Viruses though they can survive outside the animal body cannot multiply except within living cells and so in laboratories they have to be grown either in fertile living eggs or in a living culture of animal tissue This is obviously a more difficult and limiting technique than that of the culture of bacteria in a culture medium composed of broth or agar or some similar substance All animal diseases due to micro-organisms may be regarded as the reaction of a host to a parasite the animal being the host and the micro-organism the parasite The

Another factor which has profoundly affected the history of these epidemic diseases is that of the mechanism of their transmission from one host to another The mechanism of infection is sometimes a complicated one involving animal and insect intermediaries as vectors Thus the incidence of disease in man may be determined not only by the preval ence of the parasite and man s susceptibility to it but also by the ecology of the intermediate host or hosts On this line of thought there is a theory that the disappearance of plague from Europe towards the end of the seventeenth century was largely due to the replacement of the black rat by the brown and this explanation is often quoted in history books There is however one great difficulty in accepting the theory and this will be discussed later

Finally we have to consider changes in the parasite There are numerous examples of diseases which have undoubtedly changed their behaviour in a way which suggests that the causal organism has changed Sometimes the change has been a sudden one for example when influenza caused such devastation in 1918–19 or when mild smallpox first ap peared and sometimes there has been a gradual change as ex emplified by the history of scarlet fever We know little about these changes in nature but we know that in the laboratory bacteria and viruses may change their virulence consider ably Generally speaking if a bacterial culture is kept going in a laboratory for some time the organism tends to lose virulence whereas if it is pas ed through a series of animals the bacteria tend to gain virulence Some of the recent work of Burnet* on the changes in influenza virus when grown in the laboratory in fertile hen s eggs is most intriguing from the epidemiological point of view

We must remember that all bacteriological knowledge is historically new Before about 1880 the only way of classify ing diseases was by their signs and symptoms and classifica tion founded on the identification of the micro organism came late and did not always coincide with the older classi fication For this reason many chapters begin with a descrip- tion of the history of some rather vague groups of diseases

* F M Burnet 1944.

form of protective vaccine, a vaccine being a suspension of complete or part micro-organisms, alive or dead, the suspension thus containing antigens which, when inoculated, will stimulate the production of antibodies in the susceptible person Passive immunity may occur naturally, when antibodies pass from the mother to her unborn child, or it may be produced artificially by the administration of blood serum to an animal which has no antibody from one which has Passive immunity tends to be a temporary affair and in this book the words immunization and immunity may be taken to imply the active process unless the passive one is specifically mentioned

In practice there are great differences between the efficacy of different vaccines Bacteriologists are always striving to find antigens which are harmless in themselves but will produce a large amount of antibody against the particular micro-organism concerned and they have had to use many different methods to protect against different diseases Sometimes the antigen is a harmless (or almost harmless) strain of living virus for example the vaccine used to protect against smallpox sometimes it is an extract made from virulent bacteria made harmless by the addition of a chemical for example diphtheria prophylactic sometimes it is a suspension of killed bacteria, for example whooping-cough vaccine In the study of individual diseases we shall find one diphtheria whose history seems to have been affected profoundly by artificial immunization one smallpox in which the part played by immunization is difficult to assess and many whose history has so far been unaffected We have to remember that the process of natural immunization goes on all the time Sometimes it is obvious as in a community of young children which suffers an epidemic of measles and then remains free from the disease until there are enough unimmunized recruits to provide fuel for another epidemic Sometimes it is less obvious because the presence of some micro-organisms in a community may produce a great deal of immunity without producing much overt disease In this respect the history of the disease is an imperfect reflection of the reaction between the community and its parasites

I

AN EXAMINATION OF SOURCES

IT is necessary to begin with a rather protracted examination of sources because in medicine perhaps more than in any other subject ideas and nomenclature have changed enormously even in the last seventy years and almost out of recognition between 1348 and 1950

For the earliest period from 1348 to the reign of Henry VIII (1509–47) we depend for information chiefly on monastic histories state documents and for the Black Death on the rolls of manor courts There are also many fragmentary references to epidemics in letters of the time Medical books begin to be useful in the Tudor Period and become continuously more so from the time of the great physicians of the Commonwealth and Restoration – Sydenham Willis and Morton

In the Tudor Period also there were the beginnings of statistical records in the London Bills of Mortality (see Appendix p 149) and the parish registers As these will be mentioned frequently when the history of individual diseases is discussed a little more may be said about them

The London Bills of Mortality began probably in 1532 and the first of them which is extant tells how many syns the 17th day of November until the 23rd day of the same month is dead within the city and freedom young and old these many following of the plague and other diseases This Bill accounts for only sixty six deaths and is almost certainly incomplete In the next thirty years the Bills were issued irregularly probably only when plague was epidemic but between 1563 and 1592 the regular series began It is not clear how the first Bills were compiled but early in their history they became the responsibility of the Company of Parish Clerks This Company was granted a royal charter in the thirteenth century and its function was connected with

for example the continued fevers, and then branch out into a description of the diseases which probably made up that group The modern classification did not always have to wait for the discovery of the causal organism, because clinical differentiation was proceeding apace before the discovery of bacteria but bacteriology has nevertheless produced a *revolution in modes of thought about communicable diseases*

It is much easier to write the history of a disease which has characteristic signs and symptoms such as plague, than of one which has indefinite signs and symptoms such as influenza But many diseases can be identified in the ancient records from the combined study of their clinical and epidemiological characteristics

first the physicians made no use of the Bills and indeed they probably held them in contempt because they were based on the diagnoses of ignorant women It was left to a business man called John Graunt* (1620–74) to make the first serious study of the despised Bills His *Observations on the Bills of Mortality* are generally regarded as the first important contribution to the study of vital statistics ever made and his handling of the extremely inadequate material at his disposal was masterly He knew that he was on much safer ground in making deductions from total numbers of deaths than in using information about deaths from individual causes and some of the principles he adopted in analysing the Bills are still of first rate importance to anyone who tries to draw conclusions from figures of deaths by cause These principles seem simple but it is surprising how often they have been and still are forgotten When Graunt found that deaths from a certain cause seemed to be increasing in the Bills over a period of years he was always careful to scrutinize the figures of deaths from nearly related causes to make sure that deaths from the cause he was examining really had increased and that the increase was not an artefact due to a change in classification or nomenclature In plague years he found that it was not only deaths from plague which increased but also those from many other causes and he suspected that deaths from plague were being concealed because of the inconvenience which a death from plague in a house might cause to the other inmates Perhaps some of the discrepancies were due not to deliberate concealment but to genuine mistakes for a similar phenomenon is seen today in years of epidemic influenza

This seems very simple and obvious but two examples may be given to show that the importance of scrutinizing sources was and is sometimes overlooked When examining the Bills of a later period William Heberden the younger† concluded that deaths from dysentery had declined markedly He assumed that griping of the guts in the Bills was synonymous with dysentery In fact it was what we call infantile diarrhoea and the deaths from it had merely to

* John Graunt 1662 † William Heberden 1813

church music When a rich citizen died the whole company might be engaged to sing at his funeral and thus it acquired the apparently incongruous function of registering births and deaths By 1581 the procedure had become formal In every parish two *discrete matrons were selected to be searchers They were sworn before the Dean of Arches at the Church of St Mary le Bow to search the body of every such person as shall happen to die in the same parish and to report weekly* to the parish clerk The parish clerk had to make a weekly report to the warden of the parish clerks who sent it to the mayor who sent it to the minister of state The purpose of the Bills was to warn the great ones of the court and of the city when plague was about so that they might depart in good time for the purer air of the countryside

In 1625 the Company obtained a decree of Star Chamber permitting them to set up a special printing press for the issue of weekly and annual Bills At first numbers of burials from plague and from all other causes and numbers of christenings were returned In 1629 causes of death other than plague were distinguished and the totals of deaths from all causes were separated by sex *In 1727 the ages of those dying were given and in this form the Bills continued until 1849 eleven years after the establishment of the General Register Office had made them obsolete In 1955 the weekly return of the Registrar General still includes a memento of the Bills for Table III of the report is a return of the deaths by age sex and cause registered in London during the week*

For places outside London the sources of information about births deaths and sometimes causes of death are the parish registers These were kept with different degrees of care in different places from the reign of Elizabeth One of the inaccuracies of the Bills of Mortality and of the parish registers is that they did not include the deaths of Dissenters

There are then two main streams of information about the incidence and mortality of disease from the middle of the seventeenth century One is medical literature which was contributed largely by the fashionable physicians in London and makes little use of figures and the other is the incomplete information in the Bills and parish registers At

typhoid fever Deaths from diphtheria were included under scarlatina or possibly under one of the other headings for diseases of the throat until 1885 and typhoid was included with typhus until 1869 Statistics of the incidence of diseases came much later than those of deaths For some large cities the number of notifications of certain infectious diseases has been collected since 1898 but for the whole country the series began in 1911 and from time to time new diseases were added For example poliomyelitis was added in 1912 encephalitis lethargica in 1918 measles and whooping cough in 1940

The great physicians of the Commonwealth and Restoration of whom Sydenham is the outstanding example made important contributions to the clinical differentiation of diseases and so some contribution to the study of their prevalence and fatality at different times It must be remembered however that for the most part they practised in London and among the well to-do classes so that they probably knew little about the diseases of the poor which bulked so large in the Bills and even less about the diseases of the countryside In the eighteenth century the founding of voluntary hospitals and of public dispensaries brought medical men of distinction into much closer contact with the poor of the cities and some of their writings are valuable to the student of the history of diseases Towards the end of the eighteenth century fever hospitals began to be established and their records are a useful supplement to the statistics which the office of the Registrar General began to collect in 1837 One of the results of the sanitary reforms of the period 1848–60 was the establishment of local and central departments of health The first medical officer of the central department was Sir John Simon * who early set about the task of collecting and analysing information about the incidence and fatality of epidemic diseases in different parts of the country An early example of the sort of inquiry he promoted was that of Dr E H Greenhow† into deaths by cause in different districts of England and Wales From 1858

* Sir J hn Simon 1856
† F H Greenhow

be transferred to the heading convulsions The second example is rather different but also illustrates the importance of knowing one s sources It is taken from an article by Bradford Hill*

In the course of a single committee meeting I have heard a distinguished surgeon reject as quite valueless the death certificates obtained in a follow up of patients with cancer and yet express the greatest interest in the cancer mortality data by site of growth published by the Registrar General He apparently saw no inconsistency *in this attitude towards material obtained from precisely the* same sources and such an attitude is by no means rare

Up to 1839 the Bills of parish registers were the only continuous records of births and deaths but in certain places *quite good records were kept for restricted periods For* Carlisle for example there are records of deaths by age and cause for the years 1779–87 (omitting the year 1780) In 1750 Dr Thomas Short published his *New observations Natural, Moral, Civil, Political and Medical, on City Town and Country Bills of Mortality* As the title suggests this is a discursive book but it does contain many abstracts of the registers of parishes for a long period of years

In 1801 the first decennial census was taken so that for *the first time reasonably accurate and comprehensive figures* of population became available In July 1837 national registration of births and deaths began and in 1839 Samuel Farr was appointed to be compiler of abstracts at the new General Register Office For the first time comprehensive though at first incomplete information about fatal diseases became available for the country as a whole In his nomenclature of fatal diseases for statistical purposes Farr divided them into two main groups (1) Epidemic endemic and contagious diseases (2) Sporadic diseases *We are concerned* with those diseases in group 1 and Farr s list is much more intelligible to the modern reader than are the names used in the Bills The changes in nomenclature since Farr s time have been evolutionary rather than revolutionary There are two important omissions from Farr s list diphtheria and

* A. Bradford Hill, 1947

typhoid fever Deaths from diphtheria were included under scarlatina or possibly under one of the other headings for diseases of the throat until 1885 and typhoid was included with typhus until 1869 Statistics of the incidence of diseases came much later than those of deaths For some large cities the number of notifications of certain infectious diseases has been collected since 1898 but for the whole country the series began in 1911 and from time to time new diseases were added For example poliomyelitis was added in 1912 encephalitis lethargica in 1918 measles and whooping cough in 1940

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* Sir John Simon 1856
† E H Greenhow

to 1872 Simon was medical officer to the Privy Council, and during that period he promoted inquiries into the prevalence and distribution of epidemic diseases which have served as models for the many official reports since written His reports and those of his successors are necessarily the chief source from which the history of diseases in the last hundred years has been written

2

PLAGUE

PLAGUE is due to a bacillus (*Pasteurella pestis*) which can cause disease in man and in many animals among which rodents and particularly rats are the most important. The commonest form of the human disease is bubonic plague which begins as an epizootic or glandular infection among rats and reaches man through the bites of rat fleas which have deserted their normal host after its death Occasionally plague occurs in man as a deadly form of pneumonia and this form can spread directly from man to man through the air

There have been three pandemics of plague in the last 1 500 years The first was that which came with the corn ships from Egypt and Byzantium in the reign of Justinian in 542–3 the second was the Black Death which reached England in 1348 and the third began in 1894 We do not know whether the Justinian plague reached England as the chronicles of the time are not sufficiently precise to make identification of pestilences possible The Black Death is singled out by Trevelyan* as the unique disaster of English social history and after it plague persisted in England for three hundred years The latest pandemic affected England not at all in a dramatic sense but certain events probably associated with it are of great interest to the epidemiologist

The Black Death has been taken as the starting point of this book because it is one of the outstanding events of world history and because it is the first epidemic in Britain of a disease which we can identify with certainty

Nothing is known for certain of the remote place of origin of the Black Death but it seems fairly well established that the immediate source of infection for Europe was the Crimea

* G M Trevelyan 1942

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ians have concluded that probably a quarter and possibly one-third of the inhabitants of England died of plague in the years 1348–9 Some idea of the prevailing sense of desola tion and despair is contained in the following quotation from the chronicle of an Irish friar (quoted by Creighton*)

And I Friar John Clyn of the Order of Friars Minor and of the convent of Kilkenny wrote in this book those notable things which happened in my time which I saw with my eyes or which I learned from persons worthy of credit. And lest things worthy of remem brance should perish with time and fall away from the memory of those who are to come after us I seeing these many evils and the whole world lying as it were in the wicked one among the dead waiting for death till it came – as I have truly heard and examined so have I reduced these things to writing and lest the writing should perish with the writer and the work fail together with the workman I leave parchment for continuing the work if haply any man survive and any of the race of Adam escape this pestilence and continue the work which I have commenced

Most of the contemporary monastic chronicles are how ever interrupted in 1348–9 and if there is an entry under the year 1349 it consists merely of the words *magna mortal itas* One can only imagine that one chronicler was prevent ed by death from continuing his task and another could find no words to describe so terrible a calamity

The characteristic which distinguished the Black Death from later outbreaks of plague was more its universal distri bution than its high mortality in individual places It caused devastation in remote places and in the towns whereas early in its subsequent history plague became a disease particularly of the towns and of the poorer inhabitants of them London was much larger than any other town probably having a population of about forty thousand compared with the six or seven thousand of Bristol or Norwich Much of our in formation about plague after the Black Death is contained in state documents concerning the movements of the court letters of ambassadors and the like For these reasons Lon don occupies a prominent place in the history of outbreaks after the Black Death The identification of these outbreaks is

* C Creighton 1894

The eyewitnesses account given by de Mussis and summarized by Creighton* is a convincing one

The substance of this story is that the Italian merchants who were then settled in considerable numbers at the various termini or entrepôts of the overland trade from China and Central Asia by the more northern route were harassed by the Tartar hordes that they had stood a siege in Tana on the Don but had been driven out of it and had sought refuge for themselves and their merchandise within the walls of Caffa a small fortified post on the Crimean Straits (of Kertch) built by Genoese not long before that Caffa was besieged in due course by the Tartar barbarians that the investment lasted nearly three years that the merchants and others crowded into the narrow space within the walls were put to great straits and could hardly breathe being only partially relieved by the arrival of a ship with supplies that the plague broke out among the besieging Tartar host and daily destroyed thousands that the Tartars threw the pestilent dead bodies inside the walls by their engines of siege so that the infection took hold of those within the fort that the Tartars dispersed in panic and spread the infection all over the shores of the Euxine Caspian and Levant that such of the Italian traders as were able de Mussis himself with them escaped from Caffa in a ship and that the infection appeared in Genoa in its most deadly form a day or two after the arrival of the ship *although none of those on board were suffering from the plague*

Plague probably arrived in England at Weymouth (then Melcombe) at the beginning of August 1348 It is said to have *spread rapidly through Dorset Devon, and Somerset* and to have reached Bristol sometime in August From Bristol it came to Gloucester from Gloucester to Oxford and from Oxford to London, which it reached by 1 November The Eastern Counties and the north, which suffered very severely, were attacked rather later – probably in the spring and summer of 1349 By the winter of 1349 the Black Death was over but epidemic plague was established in England and was destined to recur in many lesser but still great epidemics until its final disappearance in 1665–6

It is, of course impossible to arrive at any exact figure of the total mortality from the Black Death but by piecing together scraps of information from all kinds of sources histor

* C. Creighton 1894.

The great plague of 1665 is thanks to Defoe* and Pepys† almost as well known an event in English history as the Black Death It is difficult to compare its mortality with that of previous outbreaks because we do not know for certain the number of deaths or the population Creighton‡ how ever gives the following table of comparison between the mortalities from plague in London (including the liberties and out parishes) in 1603 1625 and 1665

Year	Estimated population	Total number of deaths	Number of deaths from plague	Largest number of deaths in a week	Worst week
1603	250 000	42 940	33 347	3 385	25 Aug – 1 Sept
1625	320 000	63 001	41 313	5 205	11 Aug – 18 Aug
1665	460 000	97 306	68 596	8 297	12 Sept.–19 Sept

In the County of London today with a population of nearly 3½ million the usual number of deaths in a week in winter is round about 1 000

Although Defoe was only about six years old in 1665 and we do not know whether he was in London he used the best contemporary authorities in compiling his *Journal of the Plague Year* He wrote it in 1722 when plague had acquired a par ticular news value because of the epidemic in Marseille and Provence which was the last great outbreak in western Europe Pepys makes frequent references to plague, some of which are worth quoting because they show how the calam ity affected a distinguished civil servant

June 7th 1665 This day much against my will I did in Drury Lane see two or three houses marked with a red cross on the doors and Lord have mercy on us writ there which was a sad sight to me being the first of the kind that to my remembrance I ever saw It put me into an ill conception of myself and to my smell so that I was forced to buy some roll tobacco to smell and to chaw which took away the apprehension

June 29th Up and by water to White Hall where the court full of waggons and people ready to go out of town

* D Defoe 1722 † S Pepys 1825
‡ C. Creighton 1894

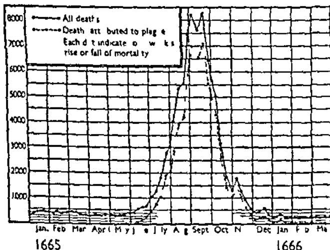
not always easy, because the word *pestis* was sometimes used in a generic sense and was not confined to bubonic plague

After the Black Death there were probably four or five considerable outbreaks of plague before the end of the fourteenth century They occurred in 1361, 1368–9, 1375 and 1390–1 There is also some record of a severe epidemic in 1381–2 which may or may not have been plague In the fifteenth century the first great outburst was in 1406–7, and there were major epidemics in London in 1464 1479 and 1500 Between these major outbreaks however there are many records of lesser ones which were sufficiently important to justify the prorogation of Parliament (for example in 1433 and 1437)

The story of plague in the sixteenth century is very like that in the fifteenth Except for a period of quiescence in the reigns of Edward VI (1547–53) and Mary (1553–8) the disease was nearly always present in London and there were great outbursts in 1500 1513 1563 1569 and 1593

The first extant Bill of Mortality is assigned with probability to the year 1532 and from 1563 one can begin to construct a continuous if incomplete picture of the annual mortalities from the disease in London Figure 2 shows the number of deaths from plague in the London Bills from 1563 to 1666 In spite of their defects the Bills give some idea of the relative importance of plague to Londoners In the four non plague years 1597–1600 the Bills showed an average of about 4 250 burials and 4 500 baptisms each year in other words an excess of births over deaths of 1,000 for the four years In 1603 plague was credited with 33 347 deaths in London (other years over 10,000) When these figures are considered in relation to one another it is easy to see why those of our ancestors who were in a position to do so moved hastily to the country when plague was about In this period also there are many accounts of plague in the provinces associated particularly with the movements of armies in the Civil War An example is the outbreak in Bristol in 1645 at the time of the siege of Prince Rupert s army by that of Fairfax It is said that there were 3 000 deaths in the city

the deaths returned in the weekly Bills from January 1665 to March 1666 Both the total number of deaths and the number of deaths from plague are shown and it is obvious that whereas at the beginning of the year some five hundred deaths were returned every week at the height of the epidemic in August and September over 8 000 a week were returned for all causes and about 7 000 from plague. It appears therefore that there may have been some 500 more deaths from plague in a week than were shown in the returns The quotation from Pepys s diary supports this view

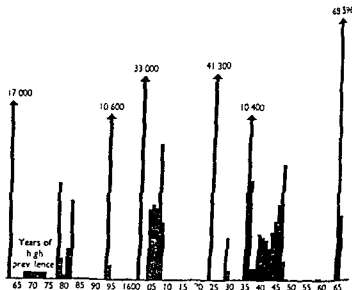


2 *Deaths from plague and other causes returned in the Bills of Mortality for 1665–6*

Figure 3 shows how the plague spread from west to east The position of the parishes (St Giles Cripplegate and Stepney) is indicated in Figure 4 which also shows how the City itself escaped relatively lightly when compared with the Liberties and Out parishes At this time the City was still the home of the prosperous merchants and the poor lived for the most part in the maze of alleys and courts outside the walls The standard of housing was worse here than in the City itself The highest mortality of all was in the populous

July 10th An unpleasing thing to be at Court, everybody being fearful one of another

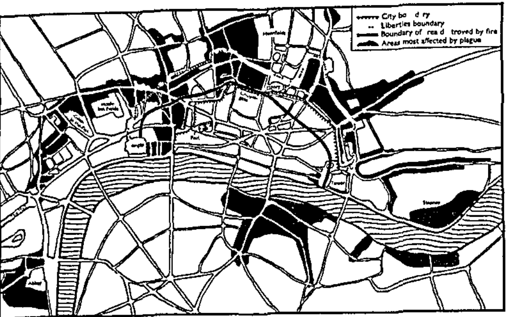
August 30th Met with Hadley our clerke for says he there died 9 this week tho I have returned but 6 which is a very ill practice and makes me think it is so in other places and therefore the plague much greater than people take it to be



1 *Annual deaths from plague recorded in the London Bills of Mortality between 1563 and 1666*

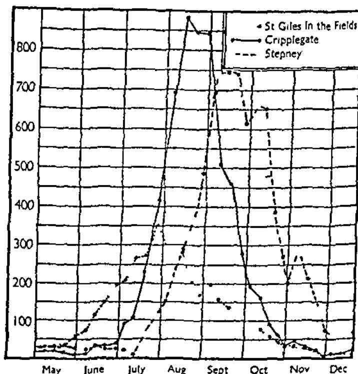
The last extract is an interesting commentary on the changes in the structure of society since Pepys's time It is difficult to imagine a casual conversation in Whitehall today between the registrar for the City of Westminster and the permanent secretary of the Admiralty in the course of which the registrar mentioned to the permanent secretary that he had just falsified his weekly return to the Registrar General!

Although statistical information about the plague of 1665 is admittedly inaccurate it does enable us to form a reasonably clear picture of the main facts about its incidence and spread Figures 1–5 illustrate the main facts Figure 2 shows



4 *Map f Lo d n showing areas affected by the plague in 1665 and dest oyed by fire in 1666*

suburbs of Cripplegate Whitechapel, Stepney, St Martin in the Fields St Giles in the Fields, Southwark, and West minster Further references will be made to this point when the possible effects of the Great Fire of 1666 are considered



3 *Chart showing trend of plague in London from west to east Based on the Bills of Mortality for 1665*

On the whole the plague of 1665-6 affected London far more than the provinces but certain provincial towns had severe outbreaks notably Yarmouth (November 1664) the eastern counties generally *(in 1665-6)* and many coast towns in Kent particularly naval stations (in 1666) There was plague also at Derby and in Derbyshire and the story of the outbreak at the little isolated village of Eyam in the Peak

beginning of January 1666 the plague was confined to two houses Four died in January eight in February six in March nine in April and only three in May On 2 June another burial occurred and then there was another pause But in a week or more the epidemic broke out with renewed power three having been buried on the 12th June three on the 15th one on the 16th three on the 17th and so on until the total for June reached nineteen The wealthier villagers at the west end had taken the alarm before and had mostly fled in the spring those who stayed kept within their houses or at least did not cross the stream Now that the infection was revived in the hot weather of June the rector s wife also proposed flight but on her husband s refusal she resolved to remain with him and to send her two children to a relative in Yorkshire At the same time the villagers in general were instinctively moved to escape from the tainted spot but Mompesson used his authority to prevent them and a boundary line was drawn round the village about half a mile in circuit and marked by various familiar objects beyond which no one was to go Mompesson s motive appears to have been to prevent the spread of the infection to the country around and his parishioners submitted passively After the end of June the villagers would have found it difficult to escape owing to the terror which the very name of their village caused in all the country round Some of them quitted their cottages and took up their abode in shelters built along the side of a rocky glen within the cordon. The Earl of Devonshire then at Chatsworth promised Mompesson that the village should not be left without supplies and people from the villages near brought their market produce to certain stated points on the boundary where the Eyam people came to fetch it the money paid being dropped into water Thus shut up in their narrow valley the villagers perished helplessly like a stricken flock of sheep By the end of June ceremonial burials came to an end the church and the churchyard were closed the dead were carried out wrapped in sheets by one of the villagers noted for his herculean strength and laid in shallow graves in the meadows or on the hillsides In July the deaths mounted up to five or six

District has become a part of English history akin to the stories of the charge of the Light Brigade or the wreck of the *Birkenhead*

The plague of Eyam is indeed the most famous of all English outbreaks Eyam was a village of some three hundred and fifty inhabitants standing among meadows around which the hills towered It had no resident doctor, but it had two ministers The one was the rector the Rev William Mompesson a young man of twenty seven, with a wife and two children who had been settled in Eyam only a year and did not like it the other was the former rector the Rev Thomas Stanley who had been ejected for nonconformity in 1662 and had remained to carry on his ministrations as a Dissenter among such of his old flock as adhered to him The wealthier householders resided at the western and higher end of the village on the other side of a brook which crossed under the road as we shall see they escaped the infection almost, if not altogether The annual village wake had been held in August 1665 with more than the usual concourse of people from villages near On 2 or 3 September a box arrived from London to the village tailor who lived in a small house at the western end of the churchyard it contained old clothes which someone in London is supposed to have bought for him cheap and some tailors patterns of cloth. This box is assumed to have been opened by one George Vicars a servant who was certainly the first victim of plague He found the contents to be damp and hung them up at the fire to dry He was quickly seized with violent sickness became delirious developed buboes in his neck and groin a plague token on his breast the third day, and died in a wretched state on 2 September His body which is said to have become putrid very soon was buried in the churchyard on the 7th Nearly a fortnight passed before another case occurred that of a youth supposed to have been the tailor s son who was buried on 22 September Before the 30th four more had died and in the course of October twenty two more were buried of plague The deaths in November declined to seven and in December they were nine There was now snow on the ground with hard frost and at the

various theories which have been put forward to explain the disappearance of plague from England

Perhaps the first chronologically is that the Great Fire of 1666 destroyed the seeds of infection so thoroughly that the disease never appeared again Defoe* ascribed this theory to certain quacking philosophers There is a modern variant of this suggestion put forward after the discovery of the mechanism of infection which postulates that after the fire there was much rebuilding in brick and stone where wood and plaster had been before and that the new brick and stone houses offered a much less favourable breeding ground for rats than did the old The insuperable objection to the theory is that it is too parochial Even if we confine our viewpoint to London we find that the area destroyed by fire was a mere fraction of the whole (Figure 4) and was the part least affected by plague Plague began and throve not in the comparatively prosperous City but in the rat ridden wooden hovels of the poor in the Liberties and Out parishes These were hardly touched by the fire of 1666 If we survey the story of plague in western Europe as a whole we find that except for a serious outbreak in Marseille and Provence in 1720–2 it had disappeared everywhere by about 1700

Creighton† himself suggested that the disappearance of plague was due to a great increase in the use of coffins for burial which prevented the contamination of the soil by plague infected bodies This idea like many of Creighton s explanations as opposed to his purely historical writing seems to have been peculiar to himself It need not perhaps be taken very seriously

A theory put forward in many books notably in Trevelyan s great *Social History* ‡ is that plague disappeared because the brown rat displaced the black. The black rat is more domesticated than the brown and is a better climber At the present day it is found in ships and in high buildings particularly in ports but elsewhere it has been wholly replaced by the brown rat Both types of rat are susceptible to plague and both harbour fleas which will attack man when their normal

* D Defoe 1722 † C. Creighton 1894.
‡ G M Trevelyan, 1942

on some days and the total for the month to fifty seven In August the dead numbered seventy-eight among them the *rector s wife on the 25th after a walk with her husband* through the meadows during which she is said to have made the ominous remark that the air smelled sweet! September added twenty four to the total, and there were now only about forty five left alive in the place Of these fifteen died up to 11 October when the mortality ceased Some of the survivors had passed through an attack of the plague among them the rector s man whose buboes suppurated Mompesson himself who had an issue open in his leg all the time escaped *the infection as well as his maid servant*

When the sickness had ceased Mompesson set about burning the infected articles in the empty cottages Three years after in 1669 he was presented to the better living of Eakring in Notts but on arriving to enter on his duties he was refused admission by the villagers and had to take up his residence in a temporary hut in Rufford Park until such time as the prejudices of his new parishioners had been overcome Between September 1665 and October 1666 259 inhabitants *died of plague at Eyam and only thirty three were* left alive Mompesson s action has often been criticized yet although the cost was terrible his action certainly saved the surrounding villages from plague

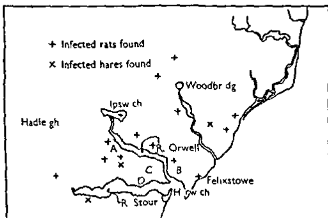
Creighton* writes the obituary of plague in Britain in the following words

Plague had been the grand infective disease of Britain from the year of the Black Death 1348–9 for more than three centuries down to 1666 The last of plague in Scotland was in 1647–8 in the west and north west of England about 1650 in Wales probably in 1636–8 in Ireland in 1650 and in all other parts of the kingdom in 1666 the absolute last of its provincial prevalence having been at Peterborough in the first months of 1667 while two or three deaths continued *to occur annually in London down to* 1679

There is an interesting but undramatic postscript to that obituary but *first it is desirable to say something of the*

* Creighton 1894

for us to consider events which are comparatively recent history and probably associated with the pandemic of plague which first became apparent at Hong Kong in 1893–4 and, like the earlier pandemics spread over most of the inhabited world though with different results from those of its predecessors



5 Plague: East Anglia 1906–18

A *F e ton 4 confirmed cases 1910 (all died)*
B *Trimley 8 suspected cases 1909–10 (5 died)*
C *Shotley 8 suspected cases 1906–7 (6 died)*
1 confirmed case 1911 (died)
D *Erwarton 2 confirmed cases 1918 (both died)*

So far as is known plague never occurred in Britain between the late seventeenth century and the beginning of the twentieth In 1900 and 1901 there were a few cases in Glasgow and in 1901 probably eleven in Liverpool and one in Cardiff All these occurred in large ports and although it was not always possible to trace the source of infection it seems reasonable to suppose that they were directly associated with rats from ships

An incident which occurred in 1910 is of more interest in that it implied a much deeper and more lasting penetration

host is dead Thus this theory depends essentially on the difference in the social habits of the two species – the black rat lives closer to man than does the brown

The great difficulty about accepting the rat theory is that of chronology Plague to all intents and purposes disappeared from England in 1666 and from the whole of western Europe at about the same time with the single exception of *the epidemic in the south of France about 1720* The earliest date given for the arrival of the brown rat in England 1688 is a legendary one for the rat is said to have arrived in the ship which brought William III from Holland In fact the brown rat probably arrived considerably later – in 1728 or 1729 according to Pennant * As late as 1783 Swaine a rat catcher (quoted by Barrett Hamilton and Hinton†), said that the black rat was the common one in London Middlesex and Buckinghamshire but that elsewhere in the Home Counties the brown had ousted it. It would be reasonable to expect a long interval of freedom from plague in London after the great outburst of 1665–6, but to fit the rat theory one must postulate an interval of a hundred years

Other theories of the disappearance of plague are more vague Liston‡ in his Milroy Lectures put forward the idea that epidemic diseases are a reflection of states of civilization He pointed out that many of the diseases which we now regard as tropical diseases for example, plague and malaria were once serious problems in Britain He ascribed the disappearance of plague to a gradual rise in the state of civilization It is difficult to prove or disprove this theory but it is doubtful whether the general state of civilization of the poor in large cities particularly in London was much higher in 1765 than in 1665 The deaths from other diseases of poverty, for example typhus rose rather than fell and this was the age of cheap gin drawn so vividly by Hogarth in Gin Lane

The conclusion must be that we do not really know why plague disappeared so suddenly when it did and it remains

* T Pennant 1776–7
† G E H Barrett Hamilton and M A C Hinton 1910–21
‡ W Glen Liston 1924

Western States of the United States there were for example six human cases in New Mexico in 1949 but there has never been a larger scale epidemic.

Although for the reasons already given we cannot ascribe the disappearance of plague from western Europe to a rising standard of civilization it does seem reasonable to attribute its recent failure to spread to the changes in standards of living since the seventeenth century

of infection In the winter of 1910–11 there occurred four cases of an extremely virulent type of pneumonia in the little village of Freston (see Figure 5) Three of these cases were in one family, and the fourth was that of a neighbour who had nursed the mother during her illness All four patients died The family doctor was puzzled by the virulence of the disease and called in a consultant physician and a pathologist from Ipswich Specimens of sputum and blood were examined and *Pasteurella pestis* was found This incident was the starting point of an exhaustive inquiry by Dr Bulstrode and other medical officers of the Local Government Board

In the light of the events at Freston certain happenings of the years immediately preceding took on a new significance It was found that in the winter 1906–7 eight cases of plague like illness with six deaths had occurred at Shotley (see Figure 5) and in 1909–10 eight cases with five deaths at Trimley It was naturally difficult to find out a great deal about these so long after the event, but there is a strong presumption that they were cases of plague The head keepers of two estates near Freston reported that from 1906 onwards rats had been dying in large numbers and similar observations relating to dead rats and rabbits were made on both sides of the River Orwell

After the cases at Freston a widespread search was made for infected rodents and the results of the search are indicated on the map The search was kept up for several years and infected rodents were occasionally found even as late as 1918 There were three more cases of human plague in the district one in a seaman at Shotley naval barracks in 1911 and two at Erwarton (see Figure 5) Nearly all the human cases occurred in poor homes with an abundance of fleas

There seems to be little doubt therefore that rodent plague was well established in this part of Suffolk from 1906 to 1918 and yet only twenty three human cases were reported in the whole period There can be little doubt that the spread of plague is very unlikely in a community with standards of living similar to those in an English rural district There is other evidence for this belief for plague is now established in the wild rodent populations of no fewer than fifteen of the

or preferably pandemics of a disease with symptoms mainly connected with the respiratory tract with an acute onset, a very high attack rate and on the whole a low case mortality Some of these characteristics are very variable For example in some epidemics the respiratory symptoms may be very obvious whilst in others they may be almost absent In some notably that of 1918–19 the total mortality has been enormous though even in that pandemic it must be remembered that the number of cases far exceeded the number of deaths In some communities in 1918–19 almost every member was attacked so that the pandemic did satisfy the criterion of a relatively low case fatality

From the historical point of view the discovery of the virus of influenza by Wilson Smith Andrewes and Laidlow in 1933 is recent and does not help in the identification of the epidemics of the past For the identification of these we have to rely on a blend of clinical and epidemiological description and must often be in doubt as to whether the disease was or was not influenza Some descriptions are however, extremely lifelike and it may be permissible to quote a particularly good one out of its strict chronological order This was written by Willis* and relates to an epidemic in 1658

About the end of April suddenly a Distemper arose as if sent by some blast of the stars which laid hold on very many together that in some towns in the space of a week above a thousand people fell sick together The particular symptom of this disease and which first invaded the sick was a troublesome cough with great spitting also a catarrh fallin down on the palat throat and nostrils also it was accompanied with a feaverish distemper joyned with heat and thirst want of appetite a spontaneous weariness and a grievous pain in the back and limbs which feaver however was more remiss in some that they could go abroad and follow their affairs in the time of their sickness but complaining in the mean time of want of strength and of languishing a loathing of food a cough and a catarrh But in some a very hot distemper plainly appeared that being thrown into bed they were troubled with burning thirst waking hoarseness and coughing almost continual sometimes there came upon this a bleed ng at the nose and in some a bloody spittle and frequently a bloody flux such as were indued with an infirm

* T Willis 1659.

3

INFLUENZA - THE SWEATING SICKNESS

INFLUENZA is caused by a virus first described by Wilson Smith Andrewes and Laidlow* in 1933

The demonstration of the existence of the influenza virus by the British workers mentioned above opened a new chapter in the study of the disease and since then an immense amount of work has been done This work on viruses is highly technical and complex and any brief description must necessarily be an over simplification Briefly there are two important influenza viruses A and B Virus A is the one usually found in major epidemics whereas B tends to occur in more localized outbreaks – in camps schools and the like The virus discovered in 1933 was A but since then many other strains of A which differ from the original and from each other have been isolated There is a general belief among those who work with influenza viruses that a succession of changes is occurring in them Strains of A isolated in different parts of the world at about the same time tend to resemble one another more than those isolated a few years earlier Even in non epidemic years some deaths are attributed to influenza but in such years a virus can rarely be isolated In order to explain this Andrewes has suggested that there may be a basic influenza virus which is not detectable by the laboratory tests at present available but which can cause illness and occasionally death In an epidemic year this virus becomes changed in some way, acquires the power of spread and can be detected by laboratory tests

It is relatively easy to write about plague because the clinical features of the disease are sufficiently characteristic to enable one to identify epidemics far back into antiquity With influenza the position is very different All that one can do is to search the records for examples of epidemics

* W Smith C.H Andrewes and P P Laidlow 1933

1528 and 1551, and then all references to the disease ceased so far as England was concerned Creighton* quotes many contemporary references to the Sweating Sickness and it seems to be reasonably well established that the disease usually occurred in the summer and autumn, attacking the rich rather than the poor often having a high mortality rate among young adults and killing very quickly There seems to be no mention of respiratory symptoms in the contemporary accounts It is hard to recognize similarities between these contemporary descriptions and influenza as we know it and the only disease of modern times which seems to have a likeness to the English Sweat is the disease described in France and known as the Picardy Sweat There is an interesting short article on Sweating Sickness by Michael Foster† in the memorial volume dedicated to Osler on the occasion of his seventieth birthday Foster was inspired to investigate the subject because he had seen a case of Picardy Sweat while serving in France in the First World War He points out that there is a marked similarity between the symptoms described by Caius – profuse sweating palpitation epigastric pain and fear of imminent death – and those described by the French writers on the Picardy Sweat The chief difference between the clinical descriptions is that the French writers describe a miliary rash which is not mentioned in the Tudor records There seems to be little doubt that the Picardy Sweat is not the same disease as influenza but whether it is the same disease as the English Sweat is more doubtful On the whole the clinical evidence is against the identification of either the English Sweat or the Picardy Sweat with influenza The relatively short space of time (1485–1551) during which epidemics of the English Sweat were occurring is roughly the same as that in which epidemics of respiratory diseases began to be reported following the pandemic of what was probably influenza in 1510 which has already been mentioned There were epidemics of acute respiratory diseases which spread over Europe in 1580–2

In the first half of the seventeenth century there were few such outbreaks but in 1658 occurred the epidemic described

* C Creighton 1894 † M C Foster 1919

body or men of a more declining age that were taken with this disease not a few died of it but the more strong and almost all of an healthful constitution recovered those who falling sick of this disease and died for the most part died by reason of the strength being leisurely wasted and a serous heap more and more gathered together in the breast with the feaver being increased, and a difficulty of breath like those sick of an hectic feaver Concerning this disease we are to enquire what procatartic cause it had that it should arise in the middle of the Spring suddenly and that the third part of makind almost should be distempered with the same in the space of a month then the signs and symptoms *being carefully collated the formal reason of this disease also its* crisis and way of cure ought to be assigned

Anyone reading that description today must feel convinced that Willis saw an epidemic of influenza

According to Thompson* the first identifiable epidemic of influenza in England was in 1510 The clinical description is similar to that of Willis already quoted, but is shorter Something is said of its origin in Malta and of its spread The epidemic spread rapidly and involved the whole of Europe According to Creighton† there is no contemporary evidence of this epidemic in English writings, but certain foreign writers stated that England was affected

One of the most intriguing epidemiological problems of these early years is that of the identity of the disease known as the Sweating Sickness or English Sweat Was it influenza or was it not? The main facts about it as given by Creighton† are as follows It appeared suddenly in 1485 either just before or soon after the battle of Bosworth (22 August 1485) It was raging in London at the end of September and in October and two mayors and four aldermen died of it There is no very detailed account of the clinical course of the disease in this epidemic but it seems to have been rapidly fatal – sometimes within six hours of onset The sweating from which the disease took its name was a prominent symptom The best known description of the disease is that of John Caius‡ and relates to the last epidemic – that of 1551 Between 1485 and 1551, there were five epidemics, in 1485 1508 1517

* T Thompson 1850 † C Creighton 1891
‡ *John Caius 1552*

Office and although the collection of information about deaths by cause was then very imperfect it is perhaps worth mentioning that in the year 1831 1 6,9 deaths from influenza were registered and 220 of these occurred in London The next explosion was in 1847–8 and in those two years some 15 000 deaths were ascribed to this cause From then to 1861 the deaths registered every year exceeded 1 000 but from 1861 to 1889 they were insignificant, and Greenwood* in his summary of the history of influenza in the Ministry of Health report on the epidemic of *1918* says *In 1889 this country had been free from pandemic influenza for more years than in any previous epoch since the middle of the seventeenth century*

The pandemic of 1889–92 was the beginning of a new chapter in the history of the disease not only in England and Wales but also in Europe and North America and probably throughout the world Since 1889 influenza has been an appreciable cause of death in many years both before and after the greatest pandemic of all time – that of 1918–19

There are conflicting statements as to the origin of the pandemic of 1889–92 but it seems to be established that Russia was affected in the early winter of 1889 Clemow† writing in the *Lancet* of 1894 produces reasonably convincing evidence of spread from western Siberia at the end of September to European Russia but the spread was extremely rapid and the disease appeared in St Petersburg (now Leningrad) in October By the end of December, most of continental Europe was affected and by the middle of December influenza was reported in New York and Boston. A few cases began to be reported in London early in December but the epidemic reached its height there about 11 January and was declining rapidly by 8 February

A problem which is always discussed during a pandemic of influenza is whether the epidemic begins in one country and spreads to others or whether it begins in several centres at about the same time In 1889–90 the disease was known in western Europe as Russian influenza , but whether the pandemic really had its origin there must be regarded as

* L.H Greenwood † D Clemow 1894.

by Willis in the passage quoted earlier in this chapter From 1661 to 1686 Sydenham* was writing his account of the seasons and reigning diseases of London and he makes many references to outbreaks of respiratory disease which may or may not have been epidemics of influenza As has been said in the previous chapter Sydenham s field of vision was some what limited as he was a fashionable physician practising in London and so did not know much of what was happening elsewhere in the country or even perhaps in the poorer parts of London itself One observation of Sydenham s has been of particular interest to epidemiologists of a later age this refers to the epidemic of 1685 in which Sydenham says that symptoms of disease of the nervous system were particularly prevalent It is not easy to decide what disease Sydenham was describing but it may have been meningococcal menin gitis or possibly encephalitis lethargica

The chief difficulty in interpreting the writings of the great clinicians of the Restoration is in deciding how wide spread were the epidemics which they described and it is in fact extremely difficult to make any estimate of the scale of epidemics until much later when national statistics began in 1837 Some attempt seems to have been made to estimate the extent of an epidemic of influenza in 1782 when the Royal College of Physicians by public advertisement asked for information from the physicians of Great Britain about the extent of the epidemic of influenza then occurring They collected information from all over the country and were able to establish that the disease started at Newcastle at the end of April 1782 and appeared in London between 12 and 18 May and in the Eastern Counties about the middle of May This seems to have been the first attempt to collect information on a national scale Throughout the eighteenth and early nineteenth centuries there were many outbreaks of influenza but it is not possible to determine whether these were pandemic on the scale of those which occurred later There is little doubt that in 1837-41 there was a widespread prevalence of influenza in Europe and England was certainly affected These were the early years of the General Register

* T Sydenham, 16-6

4

LEPROSY – THE GREAT POX (SYPHILIS) – SMALLPOX

To the modern reader leprosy syphilis and smallpox may seem to have little in common but in medieval times there was much confusion between diseases in which unpleasant lesions of the skin were found and the term leprosy in particular was probably used to describe a multitude of skin conditions Leprosy is caused by a bacillus which under the microscope looks like the tubercle bacillus The bacillus was discovered by Hansen* in 1871 Contrary to popular belief leprosy is not readily communicated from one person to another and those who contract the disease are people who have lived with lepers for many years The only reason for mentioning it in this book is the possible confusion with syphilis in medieval times Syphilis is caused by a spiral organism discovered by Schaudinn in 1905 and is communicated by intimate contact nearly always by sexual intercourse Smallpox is caused by a virus which is large as viruses go having a diameter of about 300 mμ Smallpox is one of the most infectious diseases known and it has even been suspected that the virus could be carried by the wind from a smallpox hospital to infect persons at a considerable distance from the building

LEPROSY AND SYPHILIS

It may be well to consider leprosy first as there is little to be said abou[illegible] nd from the chronological point of view [illegible] disease [illegible] tant only at the beginning of the period [illegible] b[illegible] reighton† considers that true leprosy [illegible] gland in medieval times He quotes [illegible] nglicus of which the date is un[illegible] characteristic signs and symp[illegible] ows falling bare and getting

† C. Creighton 1894

be but the temptation to speculate a little is irresistible Is it likely that a pandemic on the scale of that of 1918–19 may occur again and if so will the new drugs which have been discovered since then be so effective that the death rate will be negligible, at any rate among those in youth and early middle age? It seems probable that there will be pandemics in the future as there have been in the past and, although we shall be able to learn much more about them now that we can study the causative virus it is unlikely that we shall be able to do a great deal to prevent their spread It may be possible to prepare effective vaccines but it will not be easy to prepare them quickly enough and on a sufficiently large scale to protect whole populations

It is more difficult to make even a guess at the probable effectiveness of the new drugs such as penicillin Probably none of those known at present is effective against the influenza virus itself but we do not know whether it was the influenza virus which killed so many people in the prime of life in 1918–19 or whether the worst damage was done by secondary bacterial invaders The omens are favourable if secondary infection did the damage and less favourable if the virus itself did There is one small piece of statistical evidence recently put forward by Logan* which gives ground for a little optimism He showed that in the most recent outbreak of 1951 the proportion of the total number of deaths among persons over fifty five was no less than 88 per cent – higher than in 1933 (59 per cent) and far higher than in 1918 (14 per cent) All this is however pure speculation and all that the epidemiologist can do is to keep a weather eye open for the danger signals which heralded the pandemic of 1918 – an outbreak in the summer with a sudden age shift of mortality from old people to younger ones

* W P D Logan see W H Bradley 1951

Cordoba At about the time of the occupation of Naples from February to May 1495 the disease became widely disseminated in the army and in Italy generally and from there it spread all over Europe reaching Scotland by 1497 and London at least as early as 1503 *

The other school believes just as passionately that syphilis had occurred in Europe from time immemorial but that up to the end of the fifteenth and beginning of the sixteenth centuries it had been a less acute and fatal illness and had been confused with leprosy and many other skin diseases For some unknown reason there was at this time a pandemic of an extremely acute and fatal form of the disease which probably began in France and Spain and spread rapidly over the whole of Europe The change of type lasted some twenty or thirty years and then the disease reverted to the more chronic form of today

The first original English writer on the Morbus Gallicus or Morbus Neapolitanus as he called it was William Clowes who published his treatise in 1579 He was surgeon to St Bartholomew s Hospital and had treated many cases with inunctions of mercury His descriptions suggest that the disease had by then lost the terrible severity of the original epidemic type

The origin of the European pandemic remains a mystery and quite apart from the controversy over historical details the alternative explanations both present serious difficulties to the epidemiologist On the one hand it is difficult to believe that the disease could have travelled so far and so fast from a single place and on the other it is difficult to explain the sudden change of type if one accepts the view that syphilis had been endemic in Europe for centuries

SMALLPOX

As epidemic diseases leprosy and syphilis disappeared from the scene long ago but smallpox had a long innings and we have not seen the last of it even now The early history of smallpox in England is obscure In the medieval period

* C Creighton I (1894) p 419

knotted with uneven tuberosities the nose and other features becoming thick coarse and lumpy the face losing its mobility or play of expression the raucous voice the loss of sensibility in the hands and the ultimate break up or maufragium of the leprous growths into foul running sores It seems likely that true leprosy was declining at the end of the fourteenth century and had disappeared by the fifteenth The great interest of Creighton s chapter on the subject lies rather in the references to skin conditions which were almost certainly not leprosy at all and which may have been syphilis Creighton quotes a whole series of references from the beginning of the fourteenth century onwards to the venereal origin of leprosy and mentions an edict of Edward III to the mayor and sheriffs of London in 1346 instructing them to drive out lepers from the City One of the main reasons given is that lepers *communicate their disease by carnal intercourse with* women in stews and other secret places Some of the clinical descriptions given sound remarkably like the lesions of secondary syphilis

Whatever the lesions of the skin which were called leprosy really were there is no doubt that in the last years of the fifteenth century there was an extraordinary pandemic of syphilis in Europe If it were not for this syphilis could have had no place in this book for since then it has been an endemic and not an epidemic disease Many thousands of words have been written by medical historians about the origin of this pandemic of syphilis The controversy began early in the sixteenth century and is still unsettled One school believes passionately that syphilis was brought back from America by sailors who accompanied Columbus on his second voyage and their story runs roughly as follows Columbus left Seville on 4 August 1492 landed on Haiti and returned to Palos 500 miles from Barcelona on 15 March 1493 Many of the forty four who returned with Columbus were infected with syphilis and very soon after their return an epidemic of an extremely acute and fatal form of the disease broke out in Barcelona In 1494 Charles VIII of France was preparing for an invasion of Italy and in his army was a body of Spanish mercenaries under Gonzalo de

Cordoba At about the time of the occupation of Naples from February to May 1495 the disease became widely disseminated in the army and in Italy generally and from there it spread all over Europe reaching Scotland by 1497 and London at least as early as 1503 *

The other school believes just as passionately that syphilis had occurred in Europe from time immemorial but that up to the end of the fifteenth and beginning of the sixteenth centuries it had been a less acute and fatal illness and had been confused with leprosy and many other skin diseases For some unknown reason there was at this time a pandemic of an extremely acute and fatal form of the disease which probably began in France and Spain and spread rapidly over the whole of Europe The change of type lasted some twenty or thirty years and then the disease reverted to the more chronic form of today

The first original English writer on the Morbus Gallicus or Morbus Neapolitanus as he called it was William Clowes who published his treatise in 1579 He was surgeon to St Bartholomew s Hospital and had treated many cases with inunctions of mercury His descriptions suggest that the disease had by then lost the terrible severity of the original epidemic type

The origin of the European pandemic remains a mystery and quite apart from the controversy over historical details the alternative explanations both present serious difficulties to the epidemiologist On the one hand it is difficult to believe that the disease could have travelled so far and so fast from a single place and on the other it is difficult to explain the sudden change of type if one accepts the view that syphilis had been endemic in Europe for centuries

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there are scattered references to pockes but it is not certain whether these refer to syphilis smallpox or possibly even to other skin diseases For some mysterious reason the champions of vaccination of a later age seem to have felt that it was necessary to their argument to show that smallpox was a common and deadly disease in the Middle Ages whereas the opponents of vaccination tended to attribute these references to other diseases and to consider that smallpox was a new disease in the England of the Tudors Whatever one may think of Creighton s views on the value of vaccination one cannot help respecting him as a historian and his general account of the history of smallpox carries more conviction than does that of his opponents It is difficult to improve on Creighton s own summary

> The history of smallpox in Britain is that of a disease coming gradually into prominence and hardly attaining a leading place until the reign of James I In this respect it is unlike plague and sweating sickness both of which burst upon the country in their full strength just as both made their last show in epidemics which were as severe as any in their history

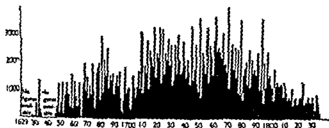
In a later passage he says

> It would be a not incorrect summary of the incidence of smallpox in Britain to say that it first left the richer classes then it left the villages then it left the provincial towns to centre itself in the capital at the same time it was leaving the age of infancy and childhood Of course it did none of these things absolutely but the movement in any one of those directions has been as obvious as in any other Measles and scarlatina have not shown the same tendency to change or limit their incidence Smallpox may have surprises in store for us but as an exotic infection its peculiar behaviour may not unreasonably be taken to mean that it is dying out – dying as in the death of some individuals gradually from the extremities to the heart.

That was written in 1894 and Creighton was convinced that vaccination was ineffective in preventing smallpox His argument that the chief determining factor in the history of smallpox has been the natural history of the disease itself unaffected by vaccination is one for which there is much

to be said but in the following pages we shall have to consider the other side of the argument and also to describe some of the surprises which smallpox had in store in 1894

The number of deaths recorded each year in the London Bills from 1729 to the beginning of death registration (1837) is shown in Figure 7a These can of course be regarded



7 (a) *Deaths from smallpox in London 1629–1837 Compiled from the Bills of Mortality*

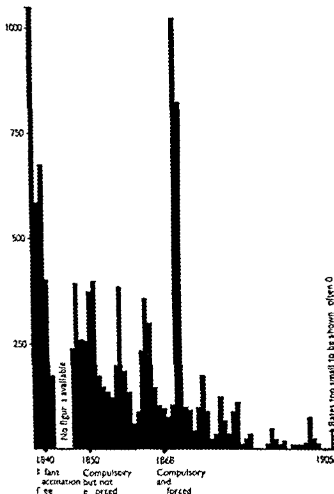
only as a very rough guide to the actual number of deaths and it must also be remembered that over this long period London was growing enormously and that the movement of population from the City into the surrounding Liberties and Out parishes was beginning From time to time the area covered by the Bills was extended but St Pancras St Marylebone Chelsea and Kensington were never included in them The Bills are a particularly inaccurate guide at the end of the period and there is a very marked discrepancy between the figure given in them for the year 1837 – 217 – and the number of deaths actually registered in the period 1 July–31 December 1837 – 762 In spite of the inaccuracies of the Bills Creighton considers that they may be taken as showing on the whole fairly the proportion of deaths from smallpox to those from other causes and the years of its greatest outbursts Figure 7b shows the annual death rates in England and Wales from smallpox from the first full year of registration (1838) to 1905 The epidemic of 1900–5 was the last considerable outbreak of major smallpox in Britain and after it the annual death rates reached a very low figure The end of major smallpox as a native disease was less

sudden than that of plague but it is probably true to say that since the time of the First World War major smallpox has been exotic It is impossible to prove this thesis because it has not always been possible to trace the source of the many small outbreaks but it seems plausible

The trend of smallpox from 1911 to 1955 is shown in the table below of notifications and deaths recorded in each year

I				II			III		
Year	Cases	Deaths		Year	Cases	Deaths	Year	Cases	Deaths
1911	295	23		1923	2485	7	1935	1	–
1912	123	9		19 4	3765	13	1936	12	–
1913	115	10		1925	5365	9	1937	3	–
1914	64	4		1926	10146	18	1938	18	–
1915	90	13	Minor Smallpox	1927	14767	47	1939	1	–
1916	149	16		1928	12420	53	1940	1	–
1917	7	3		1929	10967	39	1941	–	–
1918	63	2		1930	11839	28	1942	7	–
1919	294	24		1931	5664	9	1943	–	–
1920	263	30		1932	2039	3	1944	16	3
1921	315	5		1933	631	2	1945	4	–
1922	973	27		1934	179	6	1946	56	14
							1947	78	15
							1948	–	–
							1949	19	5
							1950	8	–
							1951	27	10
							1952	135	1
							1953	30	8
							1954	–	–
							1955	–	–

If one compares the three columns of this table it is immediately obvious that smallpox had indeed one great surprise in store when Creighton wrote of it as a dying disease in 1894 In the summer of 1919 there was a small outbreak of smallpox in Norfolk and Suffolk in which the disease had quite different clinical characteristics from those usually associated with smallpox It was far milder than that experienced in former years and was very rarely fatal. In 1920 there was another outbreak of this minor smallpox in Lancashire with 83 known cases and not a single death Throughout



7 (b) *Deaths from smallpox in England and Wales in the period from the b ginning of r gistration to 1905*

the late twenties and early thirties this form of smallpox spread widely in the midlands and north at first but in southern England and in Wales later Throughout the years of prevalence of minor smallpox there was occasional importation of major smallpox which was just as virulent as it had ever been In 1931 minor smallpox began to die out and in the year 1935 only one non fatal case of smallpox was reported for the whole country There was a small recrudescence in 1938 in which year 9 cases were of minor and 9 cases of major smallpox with three deaths In the war years importations of smallpox became less frequent and in 1941 and 1943 no cases were reported but in 1946 there were no fewer than 15 known importations which gave rise to 56 cases with 14 deaths Towards the end of 1951 minor smallpox appeared again after an interval of 13 years It went unrecognized until early in 1952 when 124 cases without deaths were reported in and around Rochdale in Lancashire. It is not certain how outbreaks of minor smallpox begin but there is strong presumptive evidence that the virus is imported in consignments of raw cotton

The present position of smallpox in England may be summarized as follows Occasional small outbreaks of major smallpox occur which can usually be traced to a vaccinated traveller returning from the East who develops a modified attack which goes unrecognized until severe secondary cases appear Minor smallpox appears at long intervals and usually in one of the cotton towns of Lancashire It tends to spread more widely than major probably because mild cases are unrecognized particularly in the early stages of an outbreak

Vaccinia (the lesions produced by vaccination) minor smallpox and major smallpox are due to infection with a virus or viruses which can be grown in fertile eggs and photographed under the electron microscope Whether all three diseases are due to variants of one virus or to different viruses is a most difficult question to answer and one may perhaps avoid it by saying 'It all depends what you mean by different and by summarizing the likenesses and differences In the laboratory material obtained from all three

diseases behaves in much the same way though there are slight differences between the appearances produced in the fertile egg by material from vaccinia and those produced by material from major or minor smallpox An attack of one disease produces strong immunity against the other two As human diseases the three are very different Vaccinia as ordinarily produced by inoculation of glycerinated lymph is nearly always a trivial affair of a small itching spot at the site of inoculation Very rarely however the rash is generalized and the illness severe Minor smallpox varies a good deal in severity but is generally speaking slightly more unpleasant than chicken pox whereas major smallpox is one of the most deadly infectious diseases known with a mortality of the order of twenty five per cent or more in the unvaccinated

One of the most difficult tasks which confronts the historian of smallpox is to form a just estimate of the part played by inoculation and vaccination in contributing to the decline of the disease The literature on the subject is enormous and much has been written both by those who regard it as heresy to question for one moment the part played by vaccination and on the other hand by those who regard vaccination as an invention of the devil It is important to distinguish between vaccination as a means of protecting an individual against smallpox and vaccination as a public health measure designed to protect a community against epidemics of the disease There can be no doubt at all that vaccination repeated at intervals of five years or so will almost certainly protect an individual from smallpox The problem is however to decide how far the limited amount of inoculation and vaccination which has been practised in England at different times has determined the trends of morbidity and mortality of the disease

Inoculation with material derived from the pocks of patients suffering from mild attacks of smallpox had been practised from very early times It was introduced into England about 1720 largely owing to the efforts of Lady Mary Wortley Montagu Throughout the eighteenth century inoculation was practised with varying frequency and varying success

Sometimes the patient had a mild attack and sometimes he had a severe or even fatal one The procedure was however too chancy to become very popular and with the introduction of vaccination in the early part of the nineteenth century it gradually fell into disuse Inoculation was made a criminal offence in 1840

In 1798 Edward Jenner published his famous *Inquiry into the Causes and Effects of the Variolae Vaccinae* His argument rested on two propositions (1) that those who had suffered from naturally acquired cowpox did not get smallpox (2) that cowpox could be carried from arm to arm and would protect all those who had been cowpoxed against smallpox. In support of his first proposition he cited some twenty instances of persons who had had cowpox and had subsequently been given an inoculation of matter from a patient with smallpox without any ill effect The intervals between the attacks of cowpox and the test inoculations of smallpox varied from fifty three years to nine months In every case the test inoculation failed to produce smallpox. In support of his second proposition Jenner cited his experiment of inoculating a child with matter from a dairymaid who had contracted natural cowpox and other children with matter from a cow suffering from the disease He did not apply the test of smallpox inoculation to all these children but to only three of them According to modern standards these experiments of Jenner s were rather thin but they did arouse a great deal of attention in London and in particular they stimulated Dr Woodville* of the Smallpox and Inoculation Hospital to undertake a clinical trial of the method Dr Woodville s experiments are of particular interest as it seems possible that some of the vaccine lymph which is in use today may have had its ultimate ancestry in them Unfortunately it is difficult to interpret the experiments Woodville got his cowpox lymph from a cow in a dairy in Gray's Inn Lane where 200 cows were kept of which some four fifths were attacked He inoculated seven persons with this material and subsequently inoculated these seven with smallpox matter at varying intervals Three of them were inoculated after an interval of

* M. Woodville 1799.

only five days The subsequent history of Woodville s experi ments is most confusing because from the original seven he inoculated a whole series of other persons with matter from the local cowpox lesions and at varying intervals afterwards gave inoculations of material from cases of smallpox In all he inoculated 200 persons in his first series and another 300 later Some of them had generalized eruptions and a few were very ill Anyone reading Woodville s own account now must be left with a strong suspicion that some of Woodville s patients had attacks of modified smallpox He expressed his conclusions as follows

Were I enabled to state a number of cases of variolous inoculation equal to those given above and reduced to a similar tabular form the comparative magnitude of the two diseases might be estimated with tolerable precision It is evident however that the matter of the vaccine disease has generally produced much fewer pustules and less indisposition than that of the smallpox for it appears from the preceding statement that about two-fifths of all the per sons inoculated for the Variolae Vaccinae had no pustules and that in not more than a fourth part of them was there experienced any perceptible disorder of the constitution But it must be acknow ledged that in several instances the cowpox has proved a very severe disease In three or four cases out of five hundred the patient has been in considerable danger and one child as I have already observed actually died under the effects of the disease Now if it be admitted that at an average one of five hundred will die of the inoculated cowpox I confess I should not be disposed to introduce this disease into the Inoculation Hospital because out of the last five thousand cases of variolous inoculation the number of deaths has not exceeded the proportion of one in six hundred But I am inclined to think that if the matter of the cowpox used for the purpose of noculation were only taken from those in whom the disease appeared in a very mild form the result would be more favou able than in the statement here given

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way produce a modified smallpox virus which by an empirical process of selection (see Woodville's last sentence) on the part of the vaccinators gradually became safer and safer The virus was kept going by arm to arm vaccination right up to 1881 when glycerinated calf lymph began to be substituted for it Arm to arm vaccination was not finally prohibited till 1898 Modern work on viruses lends some support to this theory in that in the laboratory the vaccinia virus resembles the smallpox virus rather more than the cowpox virus though the cowpox virus has a similar antigenic structure

Whatever may be the true origin of the vaccinia virus it is reasonably certain that in the early years of the nineteenth century vaccination became recognized more and more as an effective method of preventing smallpox and even more as an effective method of preventing death from smallpox The trend of deaths in London during the early years of the nineteenth century (see Figure 7a) suggests that some potent factor was at work which tended to diminish the number of deaths It is true that there were substantial outbreaks of smallpox later in the century particularly in 1871–2, and Greenwood* considered that the decline in the number of deaths in London was not a valid argument in favour of vaccination as a public health measure Creighton also pointed out that the returns of the parish clerks in the early nineteenth century were particularly inaccurate He forbore to point out however that the total number of deaths returned each year did not fall as did those from smallpox The argument that vaccination had a good deal to do with the decline of deaths is of course an argument *post hoc ergo propter hoc* but the alternative explanation that smallpox suddenly changed in type at just about the time vaccination was introduced seems more difficult to substantiate

The year 1838 was the first full year of registration of deaths in England and Wales and with all their failings the statistics available from that year onwards are incomparably more reliable and comprehensive than the earlier ones The early years of registration correspond with the last

* L H Greenwood 1935

great epidemic 1837–40 which showed smallpox in its old colours (Creighton) It is not possible to compare the numbers of deaths reported in this epidemic with those recorded in the Bills because the area included in London as a registration area was much larger than the area included in the Bills There had also been great changes in population since the eighteenth century It is however of some interest to note that the number of deaths from smallpox registered in London in 1838 (3 817) was only a little larger than the number (3 548) recorded in the Bills in 1796 This suggests a considerable reduction in mortality

From 1840 to 1870 there was a fair amount of smallpox in England and Wales but it was a mere shadow of its eighteenth century self and began to change from a disease which killed children almost exclusively to one which caused deaths among older people also In the eighteenth century some 80 per cent of the deaths were those of children under five and nearly the whole of the remaining 20 per cent of children between five and ten but in the registration period the age distribution changed at first gradually and then much more rapidly in the epidemic of 1871–2 In the period 1851–60 the deaths of children under five were 62 per cent of the total in 1861–70 54 per cent and in 1871–80 only 30 per cent Another new characteristic of smallpox in the nineteenth century was its tendency to remain an important disease in London when it was dying out in the provinces

Smallpox death- t s per million l ~ g
(Creighton (1894) Vol II p 617)

	1847–9	1850–4	1855–9	1860–4	1865–9	1870–4	1875–9	1880–4
London	460	300	237	281	276	654	292	244
Provinces	274	271	192	175	17[illegible]	339	48	34

It is not possible to give exact estimates of the amount of vaccination carried out at any of these periods but information became more accurate throughout the second half of the century as the machinery of public health administration improved Greenwood* considered that the amount of vaccination practised in London in the first twenty five years

* L H Greenwood I p 239

5

ASIATIC CHOLERA

CHOLERA is an acute infectious disease due to a micro-organism the cholera vibrio (discovered by Koch in 1883) which grows in the alimentary canal The disease is characterized by violent purging muscular cramps and rapid collapse Infection is usually waterborne

The Indian or Asiatic cholera which first showed itself on British soil in one or more houses on the Quay at Sunderland in the month of October 1831 was a new disease in a more real sense than anything in this country since the sweating sickness of 1485 *

Cholera is only a name to us in Britain now, and unless some major disaster which dislocates sanitary services over takes us it seems likely to remain so Its history is however full of interest for several rather unconnected reasons The menace of cholera was one of the most potent factors in securing the sanitary reforms which have made our towns places so different from what they were at the beginning of the nineteenth century Cholera had occurred in India before the beginning of the nineteenth century but for some mysterious reason it began about the year 1817 to spread from its native haunts not rapidly but inexorably The first stage of this first pandemic lasted until 1823 when the disease reached the confines of European Russia Between 1826 and 1838 it spread to practically the whole of the world between latitude 41 S and 65° N Thus it showed a power of dispersion almost equal to that of plague or influenza From the English point of view perhaps the most interesting speculation is why this had never happened before England had carried on an extensive trade with India for over 200 years and throughout this period London at least must have been just as favourable an environment for an epidemic of cholera

* Creighton Vol II p 793.

as it was in 1832 Perhaps a partial explanation is that as cholera has a relatively short incubation period, it could not survive the long sea voyage from India during which an epidemic on shipboard would have burned itself out The chief explanation seems to be however that in 1817 this disease of India acquired a new dynamism which carried it round the world

Conditions in all the large towns were at this time very favourable to the spread of cholera Much of the drinking water came from wells in the towns themselves which were in close proximity to cesspools Even when piped water supplies existed the water was often taken from rivers grossly polluted by human sewage In London for example some of the water companies between which there was intense competition derived their supplies direct from the Thames near Westminster No one realized that the drinking of polluted water was dangerous and indeed even at the end of the century there were still a few diehards like Creighton who refused to believe that drinking water was an important vehicle of infection Considering how favourable the conditions were cholera spread rather slowly

In Sunderland with a population of about 18 000 about 400 people were attacked in October–December 1831 and some 200 died By January 1832 the disease had gained a footing in Newcastle Gateshead North Shields and Houghton le Spring The places on Tyneside which were most severely attacked were the low lying ones near the river chiefly inhabited by poor industrial workers It was not until April 1832 that the infection began to show itself obviously in other parts of England A few suspicious cases occurred at Rotherhithe in the London Dock area in February and there was a gradual increase until mid June when the disease became more severe and affected parishes like St Giles in the Fields in the west end of London In all there were some 11 000 cases with 5 275 deaths in London in 1832 The most disastrous outbreak in the whole country was at Bilston in the Black Country in August 1832 The population was about 15 000 and in August and September there were some 3 568 cases with 742 deaths In this first epidemic it

is estimated that there were about 22 000 deaths in England and Wales which had at the time a population of about 14 000 000

England was almost free of cholera from 1832 to the second epidemic of 1848–9 but in January 1838 there was an explosive outbreak of cholera like disease in the Coventry House of Industry with 55 deaths The severity of the disease suggests that this was true Asiatic cholera

The second epidemic began in Scotland in October 1848 but did not really reach England until the end of that year In December there was a terrible outbreak in a large institution for pauper children at Tooting The school contained about 1 000 children of whom 300 had the disease and 180 died In the Spring of 1849 there was little cholera but in June the epidemic began in real earnest Once again the Black Country was severely affected as it had been in 1832 but the outbreaks in Hull and Merthyr Tydfil were even worse The epidemic chiefly affected manufacturing towns and big ports and many agricultural counties escaped altogether or had a few unimportant centres of infection In England and Wales 53 293 deaths from cholera were registered and this second epidemic was the most serious of all

The third visitation was in 1853–4 and like that of 1831 it began on Tyneside Again industrial districts were affected chiefly but some which had been severely affected in one or more of the earlier epidemics escaped Liverpool which never escaped had a moderate epidemic Merthyr Tydfil had about a quarter of the deaths experienced in 1849 but Plymouth Hull Bristol Manchester Leeds and the towns of the Black Country were more or less free of the disease

This epidemic is notable chiefly because it was the occasion of John Snow s* brilliant inquiry into the source of infection in cholera Thus thirty years before the discovery of the cholera bacillus he showed clearly that cholera was spread by water contaminated with excrement from cholera sufferers It is difficult for us now to appreciate the full measure of Snow s achievement since what he proved seems so obvious but we must remember that the climate of opinion

* John Snow 1849

before the days of bacteriology was completely different from that at the present time Then and for many years afterwards the miasmatic theory of the origin of infectious diseases was the one which was most generally believed The theory was that soil polluted with excrement or refuse of any kind gave off an atmospheric miasma which was the cause of certain epidemic diseases There was nothing specific about it and indeed it was commonly believed that one disease could change into another for example even after the clinical distinction between typhoid and typhus had been made it was commonly believed that one could change into the other The miasmatic theory was that on which the early movement for sanitary reform was based In their early years Chadwick and Simon waged war on all filth indiscriminately and regarded a heap of manure as just as dangerous as a cesspool which was contaminating a well of drinking water Snow s hypothesis that the excreta of cholera patients contained some specific poison which was transferred to the healthy by drinking water and caused cholera in them was therefore a completely revolutionary one Perhaps Snow s greatest contribution was however his use of the epidemiological method to solve the problem His inquiry of 1854 into the distribution of cases of cholera around the Broad Street Pump showed that those who had drunk the water from the pump suffered far more than those living in precisely the same conditions and geographical situation who had not drunk the water He also examined the pump itself and showed how the well had been polluted by the evacuations from sick people near by At first the General Board of Health did not accept Snow's conclusions and in a report of 1854 a committee of inquiry into the incident of the Broad Street Pump reported that We do not find it established that the water was contaminated in the manner alleged nor is there before us any sufficient evidence to show whether inhabitants of the district drinking from the well suffered in proportion more than other inhabitants of the district who drank from other sources In spite of this official scepticism the theory that water was very important in the spread of cholera received additional confirmation from differences in the

incidence of the disease in different households in south London There were two rival water companies supplying south London In 1849 both these companies took their supply direct from the grossly polluted Thames in the neighbourhood of Westminster but before 1854 company A had removed its source of supply to Thames Ditton higher up the river whereas company B retained its original source The pipes of these two companies *often interlaced in the* same street, so that there was no difference other than water supply, between most of the houses It was found that deaths from cholera were 57 1 per 1 000 houses supplied from the polluted source and 11 3 per 1,000 houses supplied from the purer source above Teddington Lock There had been no such difference in 1849 when the supplies of both companies *came from the grossly polluted source*

The last epidemic of cholera in England and Wales was in 1866 It was brought in by emigrants passing from Hull and Grimsby to Liverpool on their way to America As always Liverpool had a severe epidemic with 2 122 deaths but the only other important centres were Swansea Neath Llanelly Merthyr Chester Northwich a group of towns on the Exe, Portsmouth and certain parts of east London The outbreak in London is of particular interest in that three quarters of the deaths occurred in Whitechapel Bethnal Green Poplar Stepney Mile End, St Georges in the East and Greenwich These districts were supplied by the East London Company from the Old Ford reservoir which was grossly polluted and from which water was supplied without filtration Sir John Simon said of the outbreak of 1866 in London The area of intense cholera was almost exactly the area of this particular water supply nearly if not absolutely filling it and scarcely if at all reaching beyond it By 1866 *the main drainage scheme of the metropolis had been carried* out and all the water companies supplying the western parts had moved their sources of supply much farther up river

The history of cholera illustrates very well the difficulty of giving complete explanations of epidemiological phenomena The immediate causation of the English epidemics has been fully explained because the decline of cholera fol

lowed so closely the sanitary reforms which safeguarded water supplies from gross pollution with human sewage The problem which is still unsolved is why the disease relatively suddenly acquired in the nineteenth century an immense power of dispersion which it had apparently never shown before

6

CONTINUED FEVERS TYPHUS – ENTERIC (TYPHOID PARATYPHOID AND FOOD POISONING)

TYPHUS fever is caused by a member of a group of micro-organisms known as Rickettsia the organism lives normally in the body louse and the disease is transmitted by the bite of this insect This mode of transmission was described by Nicolle Conor and Conseil in 1911 and five years later da Roche Lima* isolated the organism (*Rickettsia Prowazeki*) The disease is characterized by severe toxaemia, a high temperature and a rash

The enteric fevers are caused by a group of bacteria (*B typhosus – B para typhosus A* and *B*) and as with typhus toxaemia a high temperature and a rash are characteristic symptoms The bacteria gain entry through food or water and live in the large intestine in consequence enteritis often with blood in the motion is an added symptom

The early histories of typhus and the enteric group of fevers are confused one with another because the clinical distinction between the two diseases was not finally settled in England until Sir William Jenner s treatise which was published in 1893 The distinction between typhus and enteric fever in the Registrar General s returns of causes of death was first made in 1869 only a short time before typhus disappeared from the English scene

In earlier times it was appreciated that the continued fevers as they are called were of different types but it was thought that one could change into the other The descriptive names for the two diseases varied from time to time but perhaps the most commonly used were those adopted by Huxham † He described a slow nervous fever' which

* H da Roche Lima 1916
† John Huxham 1755

was chiefly enteric and a putrid malignant fever which was chiefly typhus These were broad distinctions however and many other diseases were no doubt included under both headings

Although it is not possible to give any comprehensive picture of the relative importance of typhus and enteric before the middle of the nineteenth century it is possible to make reasonably intelligent guesses and to identify certain famous outbreaks which were almost certainly typhus

TYPHUS

In the Tudor Period there are at least three accounts of outbreaks of gaol fever or typhus originating in the assize courts – the Black Assizes at Cambridge (1522) at Oxford (1577) and at Exeter (1586)

At Oxford the Assizes met on 5 and 6 July 1577 and soon after the two Judges the Sheriff of the County two Knights eight Squires and Justices of the Peace and many others who had attended the court fell ill and died The symptoms were an acute and rapidly fatal delirium but no mention is made of a rash The disease spread to Merton College and also in the town but was soon over

We catch further glimpses of severe epidemics of typhus in the Civil War in both the royal and the parliamentary armies In 1643 the royal army was holding Reading and was besieged by Fairfax with an army of 15 000 foot and 3 000 horse The King and Prince Rupert failed to raise the siege but when Fairfax took the town he found it infected and his army suffered severely in the ensuing weeks so much so that he was unable to pursue his original plan of advancing on Oxford The royal army in Oxford was attacked a little later Willis * the great anatomist was a young man in Oxford at the time and he gives a clinical description of the disease which leaves little doubt that it was typhus The victims had an acute fever with delirium or stupor and a rash In 1644 there was a severe outbreak at Tiverton which had recently been occupied by both parliamentary and

royalist troops According to the parish registers 105 of the 8,000 inhabitants were buried in the single month of October 1644 and the total number of burials during the year was 443 These dramatic outbreaks were associated with the miseries of war, as typhus has so often been in human history but there is little doubt that throughout the seventeenth eighteenth, and first half of the nineteenth centuries the disease was also a constant visitor to London in particular, and, less frequently to smaller towns Throughout the eighteenth century the annual number of deaths in the London Bills from Fevers was usually about 3 000 and sometimes exceeded 7 000 On the whole these deaths slightly exceeded those from smallpox and fluctuated rather less from year to year In 1750 there was a Black Assize at the Old Bailey as a result of which there died of fever the Lord Mayor a Justice of the Common Pleas a Baron of the Exchequer, and an Alderman together with many less important persons

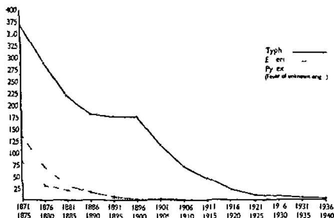
In the latter part of the eighteenth century the great growth of the manufacturing towns of the north produced conditions very favourable to the spread of typhus The poor lived in grossly overcrowded and insanitary slums and starvation was never far away It seems possible that in London typhus may have been a little on the decline Lettson * who was one of the first fashionable physicians to visit the poor of London in their homes wrote in 1773 that In the airy parts of the city and in large open streets fevers of a putrid tendency rarely arise In my practice I have attentively observed that at least forty eight out of fifty of these fevers have existed in narrow courts and alleys

Outbreaks of typhus in the nineteenth century were often associated with the great Irish immigrations which followed the potato famines In England the bad years were 1847-8 and the areas chiefly affected were Lancashire and Cheshire Liverpool being the town with highest incidence In the north western registration district of England there were 9 076 deaths in 1847 and 380 in 1848 In the latter half of the nineteenth century, typhus was declining rapidly It was

* John C. Lettson 1774.

still important as a cause of death among the poor of the great industrial towns particularly in times of industrial distress but by 1870 it had practically disappeared from southern England and lingered on only in a few of the northern ports such as Liverpool and Sunderland In the twentieth century the disease has occurred only occasionally in ports as a result of importation from abroad

Epidemic louse born typhus is perhaps the best example of a disease associated with the depths of human misery



8 *Decl ne of typhus enteric and pyrexia showing number of de ths per million of the population in Engl nd and Wales 1871–1940*

Widespread starvation and infestation with body lice are necessary conditions for the genesis of epidemic typhus but when once an epidemic has started it can spread to the more prosperous members of the community as it did at the Black Assizes of the Middle Ages (Body lice are no respecters of persons) Two modern instances may be quoted to illustrate some essential features of the epidemiology of typhus In the First World War the troops on the Western Front were nearly all infested with body lice and the louse borne disease called trench fever was common but the armies

bacteriological evidence of faecal pollution of the water supply *was produced* A primitive attempt was made to free the water mains from infection by the use of chlorine, which has since become so important a substance for the purification of water supplies

At this time the water supply of Maidstone was derived from three main sources known as the Farleigh Cossington and Boarley supplies The Farleigh supply was the most subject to human pollution and just before the epidemic started hop pickers had been camping near certain springs which contributed to this supply Most of the cases occurred in the area of the town which received the Farleigh supply and there was a number of cases in the County Asylum which also received this water The areas supplied from Cossington and Boarley had fewer cases chiefly in the later stages of the epidemic and the barracks and prison supplied from Boarley or a separate well, had no cases at all

Public water supplies have often been the source of severe outbreaks of typhoid and the most recent large scale epidemic due to this cause was that at Croydon between October 1937 and February 1938 In this period there were 310 cases and 43 deaths in a limited area of Croydon In all probability the outbreak was due to the infection of a deep well at Addington by a workman a chronic carrier, who was engaged on repair work there in October 1937 While the work was going on untreated water from the well was pumped into the public supply The cases nearly all occurred among consumers of the high level supply to which the Addington well was one of the two contributory sources The Croydon outbreak illustrates very well how a single error in the technique of safeguarding the purity of water supplies may be fatal

Water borne outbreaks of typhoid have been perhaps the largest and most explosive of any, but milk borne outbreaks are not far behind and that at Bournemouth and Poole in August and September 1936 is a good modern example In this epidemic there were 518 cases scattered throughout Bournemouth Poole and Christchurch The water supplies of the three towns were separate and inquiry

showed that all those taken ill in the first wave had drunk raw milk retailed by one firm and distributed by twelve roundsmen Immediate steps were taken to pasteurize this supply whilst attempts were made to trace the outbreak to its source Nothing suspicious was found at the headquarters of the retailing firm but at one of the small holdings (A) among the thirty seven scattered farms which supplied the retailer it was found that the farmer s wife had been taken ill on 10 August A diagnosis of typhoid fever was not made until 31 August At first this appeared to be the source case but this hypothesis did not quite fit the facts because twenty nine cases had occurred in the three towns before the farmer s wife became ill The small holding occupied by family A was part of what had originally been a farm and two cot tages In all there were five families living there A already mentioned B in the cottage next door and C D and E in the original farmhouse There had been a fatal case of typhoid fever in family B in 1934 These families were all supplied from a well which seemed reasonably satisfactory but a stream ran past them which aroused suspicion Care ful exploration of the stream above the cottages revealed that about half a mile above them there was a four inch pipe discharging opaque fluid resembling sewage It was found that this came from a large house with some sixteen occu pants the effluent from this house discharged into a small impervious tank and the overflow from this discharged via the four inch pipe into the stream Bacteriological examina tions of the effluent for the presence of typhoid bacillus in September and early October proved negative but on 16 and 24 October this organism was present in large numbers but was not found in the stream above the point of entry of the effluent One occupant of the house who stayed there at intervals and had been staying there in 1934 when the single fatal case occurred in family B was found to be a chronic intermittent typhoid carrier The carrier was stay ing in the house throughout the summer of 1936 up to 10 August when he went away and he did not return until 28 September This story has been told at some length be cause it is an interesting example of epidemiological detective

ascribed to these diseases have become so small that rates are misleading for that reason also

Paratyphoid not only is a milder disease than typhoid with a much lower mortality but also tends to differ from it epidemiologically Some of the big outbreaks of para typhoid have been like the classical milk borne epidemics of typhoid but others have been more akin to outbreaks of food poisoning due to one or other of the very many other organisms belonging to the group These organisms are widely distributed in the animal world and have variable pathogenicity for man They tend to cause gastro-enteritis of much more sudden onset and shorter duration than ty phoid In this respect, the organisms of the paratyphoid group are intermediate between typhio and the food poison ing group in that sometimes they produce a long drawn out illness known as food poisoning

Outbreaks of 'food poisoning have probably always oc curred but it seems probable that their frequency may have increased as the scale of manufacture and distribution of cooked meat and other ready cooked foods has grown In the modern world meat pies manufactured in say London may be consumed by hundreds of different people scattered all over southern England at more or less the same time Fortunately food poisoning of this kind is rarely fatal but it can occur in widespread outbreaks of sudden onset Another important vehicle of infection during and since the last war has been artificial cream In the early days of the manufac ture of this product methods of manufacture were crude and as the material was a good culture medium and the product was eaten without being cooked all the conditions necessary for widespread human infection were present In the year 1952 117 outbreaks of food poisoning affecting more than one family and probably due to bacterial organisms were reported

The epidemiology of infections with any organism of the intestinal group has some obvious links but from the his torical aspect it is perhaps the differences rather than the similarities which are interesting Cholera had a short his- tory as an important epidemic disease in England and it

would appear that in a country of western civilization a profound breakdown of sanitary arrangements would be necessary before cholera could become a serious menace again Epidemics of typhoid fever are a possible punishment for carelessness in the enforcement of precautions for the protection of water and milk supplies and outbreaks of infection with food poisoning organisms can occur as a result of some quite minor error of technique in the manufacture of a variety of food products

definitive heading of convulsions' A comparison of the weekly returns taken from Creighton for two very hot summers one at the beginning (1670) and one at the end (1718) of the period, illustrates this point

Week ending	Convulsions	Griping in guts	All causes	Convulsions	Griping in guts	All causes	Week ending
		1670			1718		
2 Aug	49	113	470	–	–	–	–
9	38	160	485	226	34	653	12 Aug
16	44	189	555	239	23	645	19
23	47	222	629	256	25	693	26
30	42	250	629	265	28	668	2 Sept
6 Sept.	31	253	617	245	27	725	9
13	24	239	586	221	26	653	16
20	38	225	575	213	27	639	23
27	27	150	474	182	24	632	30

Creighton considers that, in London diarrhoea of infants was probably at its worst in the period 1720–40 the era of cheap gin In any case there can be no doubt that in every hot summer for at least 300 years before 1911 there was an enormous mortality among infants from diarrhoea

At the beginning of the era of comprehensive statistics from 1837 onward, it became obvious that the great manufacturing towns of the north suffered even more severely in epidemics of summer diarrhoea than did London The towns where many women were employed in industry were particularly bad The only real change which occurred in the nineteenth century was that mortality fell slightly in London whereas in the great manufacturing towns it tended to rise a little

In the year 1911 no less than 31,900 deaths among infants under a year were ascribed to diarrhoea and enteritis, and in 1956 only 319 were reported Besides this enormous decline in the absolute number of deaths there has been a change in the seasonal distribution In 1911 nearly all deaths from diarrhoea and enteritis in infants occurred in the late summer and early autumn whereas nowadays the deaths from this cause are much more evenly distributed throughout the year

Nowadays fatal diarrhoea tends to occur in outbreaks in institutions where a number of young babies are congre gated A great deal of work has been done on the bacteriology of these outbreaks and the general conclusion seems to be that they are generally but not always due to particular kinds of bacillus coli The subject is a difficult one because other kinds of bacillus coli are normal and harmless in habitants of the human intestine It seems almost as though a disease which now occurs only as an occasional if dangerous visitor in institutions was formerly a nation wide scourge in every hot summer It is interesting to speculate on possible explanations for this one which has been suggested is that the decline in summer diarrhoea may have been associated with the decline in the use of horses This theory involves the assumption that the disease was largely fly borne and that the disappearance of the heaps of manure a splendid breeding ground for flies from our cities so reduced the fly population that the great summer epidemics ceased Per haps all one can say is that the theory seems plausible but cannot now be proved or disproved

Death rates are impersonal things and it may be helpful to summarize the history of fatal diarrhoea in infants under one year in England and Wales by giving total numbers of deaths in some representative hot summers

Year	Deaths from diarrhoea	Deaths from all causes	Ratio between deaths from diarrhoea and deaths from all causes
1911	31 900	114 600	1 3 6
1921	11 705	70 250	1 6
1933	3 558	36 960	1 10
1949	1 963	23 882	1 12

Thus the number of deaths in 1911 from diarrhoea alone was 8 000 more than the deaths from all causes in 1949 Moreover the proportion of infantile deaths caused by diarrhoea has decreased from 1 in 3 6 in 1911 to 1 in 12 in 1949

may have been describing diphtheria as well Huxham's description also seems to refer to severe scarlet fever rather than to diphtheria Both Fothergill and Huxham make frequent reference to a skin rash and Huxham speaks of generalized dropsy

At the end of the eighteenth century Willan* gave a remarkably good and complete description of scarlet fever in his book on cutaneous diseases He described three types

1 Scarlatina simplex with a rash and no sore throat
2 Scarlatina anginosa with a rash and a sore throat which might be very severe
3 Scarlatina maligna which was a very severe form of scarlatina anginosa with sloughing of the soft tissues of the throat and mouth and profound toxaemia

He pointed out that cases of very different degrees of severity could occur in the same household and that the sore throat occurred without the rash He states that the severe form of the disease was the leading epidemic disease in London in 1786 From 1804 to 1816 Bateman† kept records of the diseases of London based on his experience as physician to the Public Dispensary and consulting physician to the Fever Institution in London In Autumn 1807 and again in 1808 he wrote that scarlatina was generally mild presenting the eruption with a slight sore throat In the autumn of 1814 he wrote as follows

> The scarlatina has been in all cases accompanied by sore throat in the adults indeed in two or three families the throat as is usual was the only seat of the disease as the rash did not appear on the skin. Under the cool treatment which however is often imperfectly accomplished in the close and crowded apartments of the poor these cases have uniformly done well

Bateman also mentions dropsy following scarlet fever

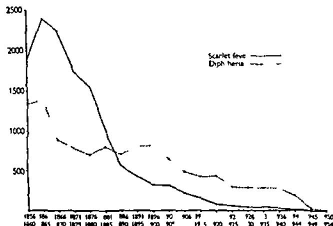
In the light of our modern knowledge of streptococcal sore throat is seems clear that bad epidemics occurred in the eighteenth century that there were quite long intervals between them and that sometimes in the early years of the

* R Willan 1798–1808 † Thomas Bateman 1819

nineteenth century – probably about 1803 – the disease became milder though it was still common

NINETEENTH AND TWENTIETH CENTURIES

The mild phase of scarlet fever lasted until about 1830 but from then on the disease began to increase in severity again and in the first two and a half years (1836–9) of registration the number of deaths attributed to this cause was considerable In 1840 the number of deaths nearly doubled and for



11 *Deaths from scarlet fever and d phtheria per mill on l v g under fifteen years of age 1856–1954*

a generation after scarlet fever was the leading cause of death amongst the infectious diseases of childhood Creighton says of this period The enormous number of deaths from scarlatina during some thirty or forty years in the middle of the nineteenth century will appear in history as one of the most remarkable things in our epidemiology The year of highest mortality was 1863 when the death rate of children under fifteen was 3 966 per million living In 1863 there were just over 30 000 deaths from scarlet fever and in 1953 there were 27

Figure 11 shows the trend of death rates from scarlet fever of children under fifteen from the time when scarlet fever and diphtheria were separated in the statistical returns to the present The marked decline of mortality began in the quinquennium 1866–70 and has continued pretty steadily ever since No satisfactory explanation of this decline has ever been offered but it may be due to a diminution of virulence of the haemolytic streptococcus as a similar fall has occurred in other streptococcal diseases such as erysipelas and puerperal fever and has also affected acute rheumatism which is usually an indirect result of streptococcal infection It seems unlikely that advances in medical treatment have had much to do with this decline because the change set in long before the sulpha drugs and penicillin were discovered It is perhaps a little ironical yet deserving of thought that penicillin which is so potent a remedy for streptococcal diseases should have been discovered after the streptococcus had lost much of its virulence Epidemiologists with a taste for history have often sounded a note of warning about the possible future trends of streptococcal diseases They have pointed out that scarlet fever varied a great deal in severity before the middle of the nineteenth century and that it may again become a deadly disease. Against these gloomy forebodings may be set the fact that the decline in severity has gone on steadily for nearly a century and that we now have drugs which will kill the streptococci without hurting the patient

DIPHTHERIA

EARLY HISTORY

Some examples of outbreaks of severe sore throat occurring in the eighteenth century have been given already Most of the accounts quoted have suggested that the disease described was scarlet fever rather than diphtheria but there are others in which the description is more like diphtheria than scarlet fever

In 1750 John Starr of Liskeard in Cornwall described an outbreak of severe sore throat in which the prominent sym

toms and signs were agonized breathing and an inflammation of the throat with the formation of membrane In one case that of a child the father pulled from the child s mouth a complete membraneous cast of the wind pipe

Although Huxham s* descriptions of the disease in Plymouth about 1751 seem to refer to severe scarlet fever he also mentions membrane in the wind pipe which suggests diphtheria rather than scarlet fever All one can say about this difficult early period is that the evidence seems to suggest that severe scarlet fever was a more prevalent disease than diphtheria but that diphtheria did occur

It seems certain that diphtheria was not an important disease in England in the first half of the nineteenth century though there were probably small outbreaks e g Ashford (Kent) 1817 Lifton (Devon) 1852

In France however it was important and the modern description of the disease was given by Bretonneau of Tours in 1821 in a paper to the Academy

MODERN HISTORY

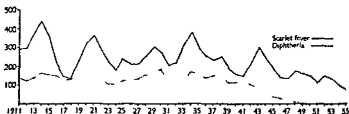
The modern history of diphtheria in England begins with the wave of prevalence which started about 1855 Simon s† second report to the Privy Council for the year 1859 has a section of 160 pages about it He says that the disease was almost unknown to British doctors until 1855 but in the four years since then it had become widespread As is usual when an epidemic disease which has been rare becomes common importation from abroad was blamed and at first diphtheria was known as the Boulogne sore throat If it was indeed imported it seems to have spread far and fast In 1855–6 epidemics were reported from places so widely scattered as Launceston (Cornwall) Spalding (Lincolnshire) Thame (Oxfordshire) Billericay (Essex) Ash (Kent) and many other places In 1859 the 9 587 deaths ascribed to the disease were scattered throughout England and Wales

At first diphtheria was overshadowed by scarlet fever as a cause of death among children but in the quinquennium

* J Huxham 1755 † Sir John Simon 1860

1886–90 it took the lead (see Figure 11) and remained a leading cause of death in children up to 1941

When epidemic diphtheria first appeared it tended to affect rural areas more than urban, but in the second half of the nineteenth century and the first forty years of the twentieth there was a gradual change so that in the decennium 1871–80 the rural areas had a death rate only slightly higher than that of the urban and by 1939 the County Boroughs had a substantially higher rate than the Rural Districts In the early years of the twentieth century diphtheria tended to kill children under school age more than



12 *Notifications of scarlet fever and diphtheria in England and Wales per 100 000 of the population 1911–55*

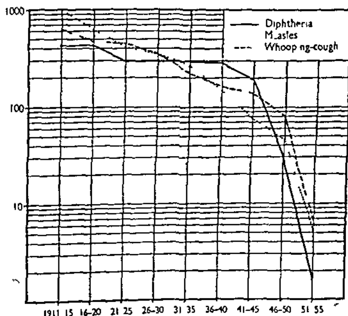
older children but here again there was a gradual change and in 1938 diphtheria was the chief cause of death among children aged 5–9

The trend of mortality among children is shown in Figure 11 After the first outburst in the sixties the death rate remained more or less stable at about 800 per million living until about 1900 then there was a slow fall to about 300 in 1921–5 and thereafter a period of stability until 1940 Antitoxin was first used in treatment in England in 1895 and it seems reasonable to suppose that the reduction in mortality between 1896–1900 and 1921–5 was chiefly due to the improvement in the treatment of the established disease More efficient treatment saved many lives but it had no effect on incidence and the graph of annual notifications (Figure 12) showed fluctuations but no consistent downward trend The diphtheria bacillus itself lives chiefly

in the throat and nose of the host causing a local inflammation but it also produces a powerful poison (toxin) which circulates in the host's blood and causes the more serious symptoms and signs of diphtheria The antitoxin which is so effective in the treatment of established disease is an agent for passive immunization and can be used to secure temporary protection against catching the disease It is made by the active immunization of horses and the serum from the immunized horse which is rich in antibodies is injected into the person to be protected Passive immunization is only temporary and clearly some safe method of producing the active immunization of man was always likely to be far more effective in controlling the disease than passive immunization Bacteriologists recognized this quite early and in 1913 the first effective diphtheria prophylactic was produced Unfortunately this was not absolutely safe and it was not until 1923 that an effective and safe antigen for the protection of man was produced In the late twenties and the thirties a certain amount of active immunization of children was done in Britain but was insufficient to influence diphtheria as a disease of the community In other countries however notably in the United States and Canada a great deal was done and it became clear that active immunization of large numbers of children could reduce the incidence of and mortality from diphtheria in the community In 1940 the Ministry of Health inspired by Sir William Jameson its chief medical officer embarked on a campaign to popularize the immunization of all children against diphtheria The mortality from diphtheria from 1911 to 1955 is shown in Figure 13 in which it is compared with those of measles and whooping-cough

In 1941 an extremely rapid decline of both incidence and mortality of diphtheria began and has continued until the present time In a dozen years diphtheria has become a very rare disease so much so that a generation of doctors is growing up who have never seen a patient suffering from it. The recent history of diphtheria in the countries of Western Europe has been quite different Virulent diphtheria came with the German armies and in the occupied countries

there was a steep rise in the death rate from diphtheria during the years of the Second World War In Holland for example the death rate rose from 0 9 per 100 000 living in 1939 to 46 per 100 000 in 1945 Since then it has declined In 1945 there were 4 270 deaths and in 1952 only 173



13 *Decline of mortality in England and Wales from diphtheria measles and whooping-cough 1911–55*

Memories are short and there is no doubt that it is now very much more difficult to persuade parents to have their children immunized than it was in 1941 This is a disturbing state of affairs but the wholesale immunization of children has had one unexpected result which makes it a little less disturbing than it might at first sight appear to be In theory there is no reason why immunization against the toxin of the diphtheria bacillus should have any effect on the population of diphtheria bacilli One might reasonably expect

that the number of healthy human carriers would increase as the number of cases of overt disease diminished There is some evidence however that in fact the population of diphtheria bacilli has declined *pari passu* with the decline of the disease Small epidemics of diphtheria do however still occur and are particularly dangerous because the disease is now so uncommon that it is often some time before a correct diagnosis is made

Diphtheria is the most convincing example of a disease which can be controlled by artificial active immunization and any reasonable person must be convinced that in England the hard work of public health officers has been the most important factor in determining its virtual disappearance in the last twelve years Their achievement has been considerable In 1941 there were 50 000 notified cases with 1 622 deaths and in 1956 there were 37 cases with 6 deaths

9

MEASLES AND WHOOPING COUGH

MEASLES and whooping cough are not particularly alike in their symptoms but they have other affinities which justify their sharing of a chapter Perhaps the most obvious of these is that nearly all of us have at some time in our lives had both diseases and it seems likely that most of our ancestors had much the same experience From the notification returns it would appear that measles is about three times as common as whooping cough but whooping cough is more difficult to diagnose because the characteristic 'whoop is not a constant symptom, and so the returns are less complete than are those of measles Both diseases still have their dangers for young children though they have become much less dangerous in the twentieth century In both the complications rather than the original diseases cause death and disability

Measles is caused by a virus but little is known of its size or shape It has been cultivated in tissue from a chick embryo and several species of monkey can be infected with it In contrast with some other viruses for example that of influenza, the virus of measles appears to have remained stable for hundreds of years When a susceptible person is exposed to infection he almost invariably develops measles and the resulting immunity is nearly always complete and long lasting Because so many people have had an attack of measles serum prepared from an ordinary blood bank is rich in antibodies and this property has been used to modify the attack in weakly children after a known exposure to infection Nowadays the usual procedure is to give an injection of γ globulin (that fraction of the serum protein which contains the antibodies) to the exposed child about five days after exposure The object is to produce a modified attack which will save the weakly child from a severe but will at the same time produce lasting immunity

Whooping-cough is caused by a small bacillus discovered by Bordet and Gengou* in 1906 An attack of the disease usually produces lasting immunity but second attacks are not quite so rare as are those of measles Many attempts have been made to produce a safe and effective vaccine to protect against whooping-cough, and modern vaccines will produce immunity in a good proportion of children

On the whole the bacteriology of these two diseases is not important to an understanding of their history The diagnosis depends and always has depended chiefly on signs and symptoms and these are sufficiently characteristic to enable us to trace their history reasonably well Measles is a febrile illness which starts like a severe cold and has a blotchy rash which comes out on the third or fourth day In whooping cough the characteristic is the cough which becomes more and more paroxysmal each paroxysm ending with the characteristic crowing inspiration or whoop

MEASLES

EARLY HISTORY

The name mezils seems to have been used first by John of Gaddesden (1280–1361) who applied it both to measles and to certain leprous lesions In the Arabic writings variolae (smallpox) and morbilli (measles) are an inseparable pair and the same is true of smallpox and mezils in the writings of Tudor times At that time measles was generally thought to be a mild form of smallpox though the severe form of haemorrhagic smallpox was also confused with it Probably by 1620 when the Parish Clerks first returned measles under a separate heading in the Bills the distinction between smallpox and measles was reasonably well understood In the first series of London Bills from 1629 to 1660 the deaths vary from only 2 in 1630 to 153 in 1656 In the next series the numbers of deaths became greater 311 in 1664 and 295 in 1670 The epidemic of 1670 is notable because it gave Sydenham† his opportunity and his clinical description would seem quite at home in a modern textbook There was

* J Bordet and O Geng u 1906 † T Sydenham 16 6

another bad epidemic in 1674 with 795 deaths in the Bill and then a long interval of low mortality until 1705–6 whe nearly 800 deaths were returned for the two years In th early period the bad epidemics apparently came at lon intervals In the eighteenth century epidemics in Londo became more frequent and by the middle of it were occur ring every other year or every three years but not with th biennial regularity which was so prominent a feature c measles in London in the nineteenth century and the earl years of the twentieth

In the early years of the nineteenth century smallpo began to decline as a common cause of death in children and measles and whooping-cough became more prominent though it is difficult to say whether the number of death increased absolutely or only relatively In this period im mediately before the introduction of universal registratio of births and deaths the Bills are notoriously incomplete In the first four complete years of registration 1838–42 the common infectious diseases of childhood had the following mean annual death rates per million living at all ages

Scarlet fever	797
Measles	539
Whooping-cough	504

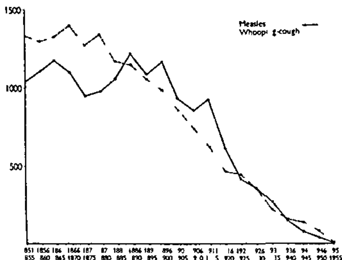
Diphtheria had not yet been separated from scarlet fever but as we have already seen it seems unlikely that it was an important cause of death at this time Scarlet fever was just beginning to assume the prominence which became obvious in the sixties and seventies and measles and whooping-cough had rates about the same as they had in the second half of the century

MODERN HISTORY

The trend of the death rate of children under fifteen from 1851 to 1955 is shown in Figure 14 At the beginning of the period measles had a slightly higher rate than that of whooping-cough but the rate for measles began to decline later and even in the early years of the First World War it was very nearly the same as it had been in 1871–5 Sin

1915 in which year there was a bad epidemic there has been a rapid fall in mortality In 1915 16,445 deaths resulted from measles and in 1955 only 176 in spite of the fact that there were nearly half a million notifications (the disease was not notifiable in 1915)

There is no evidence that the incidence of measles has decreased at all It has been generally notifiable only since 1940 and the annual notification rates since then are shown



14 *Deaths of children in England and Wales under fifteen from measles and whooping-cough per million of the population 1851–1955*

in Figure 15 Incidence varies enormously in different years for example there were about 160 000 notifications in 1946 and about 600 000 in 1951 The reduction in mortality has therefore been entirely due to the fact that the disease has become less fatal than it used to be

The reasons for the decline of the fatality of measles are probably complex It began to fall many years before there were any really effective drugs for the treatment of the fatal complications Deaths from measles have always tended to

occur in young children particularly in those between six months and two years old Those under six months have antibodies derived from their mother and so are less liable to develop the disease than slightly older children and children over two have a stronger hold on life than the very young Anything therefore, which will tend to postpone the age of attack will tend to make the disease less mortal It seems likely that much of the reduction from about 1915 to about 1935 was due to the reduction in the size of families particularly of poor families This favourable influence oper



15 *Notifications of cases of measles and whooping cough in England and Wales per 100 000 of the population 1940–55*

ated in two ways The older children who go to school pick up infectious diseases there and bring them back to their younger brothers and sisters When there are many younger brothers and sisters in the population measles will tend to occur at an early age and will tend to be more dangerous In addition to the danger of infection at an early age in the large family there is the obvious factor of poor feeding and all the other factors which make up the complex of poor social conditions The total effect of these influences on mortality from measles was examined in the Registrar General's Decennial Supplement 1931, p 167 The mortality from measles of children aged one in social class V was

nearly twenty times that of children of the same age in social class I

There is no convincing evidence in the national figures indicating that the sulpha drugs and penicillin have had much influence on mortality The decline has been a little steeper since about 1935 than it was before but not very markedly so Passive immunization with serum or γ globulin has never been used on a sufficient scale to have any influence on national figures.

In contrast to diphtheria measles is a disease in which changes in social conditions rather than advances in methods of prevention or treatment have been the important factor in determining the decline of mortality The decline of virul ence of the haemolytic streptococcus which was discussed in the chapter on scarlet fever has also had something to do with it because infections with haemolytic streptococci used to be common and fatal complications of the disease

WHOOPING COUGH

Creighton* says that there is singularly little reference to whooping cough in early medical writings and suggests that a possible reason may be that it was generally left to the management of parents and nurses and also that it may have been thought of as a complication of other diseases rather than as a disease in its own right There is however a refer ence to the kink in a medieval prescription book The first medical description of an epidemic was that given by Baillou† of Paris in 1578 Sydenham‡ mentions it under the name pertussis which we call hooping cough but always in as sociation with another disease – in one instance with measles and in another with influenza There is one description written by a mother to her husband in 1661 which will arouse a little wave of sympathy in the heart of any modern parent who has nursed a child through the disease I am in a sad condition for my poor children who are all so trobled with the chincofe that I am afraid it will kill them There is many dye out in this town and many abroad that we heare of I am fane to have a candell stand by me to goo

* C. Creighton 1894. † Baillou of Paris 1578
‡ Sydenham 1676

in too them when the fitt comes' In a later letter Moll is much the worst They have such fits that it stopes theare wind and puts me to such fits and feares that I am not myselfe In a third letter the children are said to be better and the mother asks her husband to bring a paper of lozenges for them It seems clear that in the seventeenth century whooping cough was a common and distressing malady of children just as it is now, but as a cause of death it was overshadowed by other diseases, particularly by small pox and so did not attract much notice from doctors

In the Bills of Mortality hooping cough and chincough' do not achieve a separate heading until 1701 and for fifteen years the deaths returned are only units in 1715 they rise to tens and by the end of the century are counted in hundreds – in 1780 for example 573 deaths were ascribed to hooping cough It seems probable that in the early part of the century many deaths really due to whooping cough were returned under the indefinite heading of convulsions

In the early years of the nineteenth century measles and whooping cough together began to replace smallpox as the principal killing disease of young children In Glasgow, Robert Watt* demonstrated this by a painstaking examination of the Parish Registers and published his findings as an appendix to his *Treatise on the History, Nature, and Treatment of Chincough* Although in most years smallpox killed fewer children than measles and whooping cough it still surpassed them from time to time when it became epidemic In the first six months of registration in England and Wales the deaths from the principal causes of death among children were as follows

Convulsions	10 729
Smallpox	5 811
Measles	4 732
Whooping-cough	3 004
Scarlatina	2 550

An epidemic of smallpox was just beginning at this time but had not reached its full force in London In London in contrast with the rest of England and Wales the number

* Robert Watt 1813

of deaths from measles and from whooping-cough separately greatly exceeded the number of deaths from smallpox

The trend of mortality from whooping cough in children under fifteen from 1851 to 1955 is shown in Figure 14 From 1851 to 1880 it altered little and was rather higher than that of measles but it began to decline rather earlier than did the death rate of measles and in the quinquennium 1886–90 it fell below that of measles and remained below until the quinquennium 1921–5 Since then now one disease and now the other has been the more important their relative importance depending chiefly on incidence

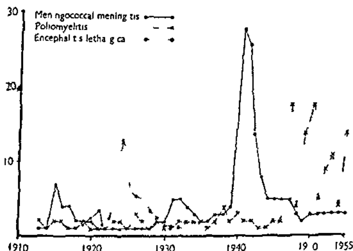
Of late years the death rate of whooping-cough has been consistently higher than that of measles but in 1955 it was the same that is 24 per million living under 15 The actual number of deaths attributed to measles in 1955 was 176 and to whooping-cough 88 but a higher proportion of the deaths from whooping cough occurred in infants under a year old

The same influences which have led to the decline of mortality from measles have resulted in the decline of mortality from whooping cough As we have seen mortality from whooping cough began to fall rather earlier than that of measles and it may be that this is due to the fact that whooping-cough tends to have its highest mortality at a rather younger age than measles In 1931 mortality from whooping cough of babies one year old was very much higher in children of social class V (i e the R G s lowest social grade) The difference was nearly but not quite as great as in the case of measles It would be interesting to be able to trace the history of the case fatality (deaths and notifications) of these two diseases further back than 1940 but unfortunately they were not generally notifiable before then In 1940 one in every seventy cases of whooping cough died In 1955 only one death occurred for every nine hundred cases The recent history of death rates for whooping cough and measles (Figure 14) shows that the trend in recent years has been similar for both diseases The fall has been particularly steep since 1947 and this may have been due to the more effective treatment of complications by penicillin and other antibiotics There is no sign in the

for regarding these fevers as any one of a number of other diseases with typhus perhaps the most probable

On the whole, therefore it is an interesting but unprofitable exercise to spend much time on the early history of the epidemic diseases of the central nervous system, and in the study of individual diseases the starting point will be the first modern description

From time to time there has been confusion between the three diseases, and indeed between them and other diseases



16 *Notifications of epidemic diseases of the central nervous sy tem in Engla d and Wales per 100 000 of the population 1913–55*

For example when widespread epidemics of poliomyelitis began in 1910 and 1911 they were at first mistaken for epidemics of cerebrospinal fever and when encephalitis lethargica appeared in 1918 it was mistaken for a severe form of food poisoning known as botulism which also affects the central nervous system Clear cut cases of these diseases are not difficult to distinguish one from another but there are many atypical cases in which it is difficult to make an exact diagnosis For these reasons statistics are inevitably inexact They do however give a general picture

of incidence and mortality which is reasonably accurate (Figure 56)

In all three diseases the number of persons who become infected with the causal organism far exceeds the number who develop obvious disease The bacteriologists have shown that this is true of cerebrospinal fever and poliomyelitis but as the organism which causes encephalitis lethargica has never been directly demonstrated the theory that healthy carriers are important rests only on analogy with the other diseases Healthy carriers and patients with mild and atypical disease are of course important reservoirs of infection in many epidemic diseases but they are particularly important in the epidemic diseases of the central nervous system

CEREBROSPINAL FEVER

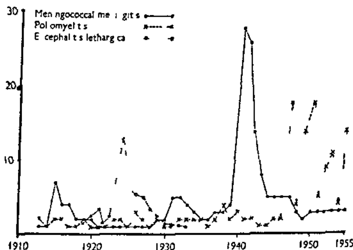
Cerebrospinal fever (spotted fever meningococcal meningitis) is an acute inflammation of the membraneous coverings of the brain caused by a bean shaped organism which is usually found in pairs within the cells in the cerebrospinal fluid The meningococcus was discovered by Weichselbaum* in 1887 but the disease had been distinguished clinically long before though it may have been confused with meningitis due to other organisms The main symptoms and signs are intense headache vomiting high temperature and stiffness of the neck so severe that the head becomes bent backwards by the spasm of the muscles and is held in that position These are signs and symptoms of any form of acute meningitis and the final diagnosis depends on examination of the cerebrospinal fluid It so happens however that epidemics of very acute meningitis are always due to the meningococcus and we can assume that those described before bacteriological confirmation was possible were cerebrospinal fever The meningococcus is found in the secretions from the nose of perfectly healthy people particularly in times of epidemics and it seems that the disease cerebrospinal fever is a rare manifestation of infection with the meningococcus

* A. Weichselbaum 1887

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and there was a rapid falling off in subsequent years so that by 1922 annual notifications had reverted to the usual endemic level In this epidemic the proportion of cases was greater in the services than in the civilian population and the disease was commoner among recruits than among seasoned troops Glover and others showed that in overcrowded barracks where cases of cerebrospinal fever occurred the appearance of obvious cases of disease was preceded by a rise in the number of healthy carriers of the meningococcus Sometimes as many as 60 per cent of the troops in a barracks might be harbouring meningococci in the nose and throat whereas the incidence of obvious disease was under 1 per cent

Between the World Wars the level of incidence remained fairly constant except for a period of activity in the years 1931–3 but in the first winter (1939–40) of the Second World War there was a recrudescence of activity very like that of the first but on a larger scale In the year 1940 notifications were 11 185 for civilians and 1 586 for non civilians

The chief differences between the epidemics of the two World Wars were that in the first it was the services which were chiefly affected whereas in the second it was the civilian population and in the second War the fatality of the disease was much less than it had been because the sulpha drugs which had come into general use about 1938 were effective in treatment It is impossible to be quite sure that the epidemic of 1940–3 was much larger than that of 1915–18 because it is likely that ascertainment was more complete in the later period The difference between the number of cases in the two epidemics is however considerable and is probably too big to be accounted for purely by better diagnosis In the epidemic of 1914–18 there were about 10 700 cases in England and Wales whereas in that of 1939–43 there were about 34 700 but in the earlier epidemic 4 136 deaths were among civilians whereas in the later one the number of deaths was 6 916 representing a much lower case fatality – 20 per cent against 39 per cent In recent years when meningococcal infections have been endemic rather

The first identifiable description of an epidemic was given by Viesseux* and related to an epidemic at Geneva in 1805 though as has been said, it is not certain whether or no some of the 'nervous' fevers described by the physicians of the Restoration period may have been cerebrospinal fever In the early stages of its history it was noticed that the disease tended to be associated with troops living in over crowded barracks There were several examples of this among the French garrisons in the Napoleonic wars Hirsch† writing in 1886 divided the history of the disease in the world as a whole into four periods (1) 1805–30, (2) 1837–50 (3) 1854–75 (4) 1876–86 but apart from two rather doubtful small epidemics in England one on Dartmoor in 1807, and one at Sunderland in 1830 it did not affect that country until 1865 and even then not very much In writing about this period Simon medical officer of the Privy Council, describes cerebrospinal fever as a disease which we in England had hitherto hardly heard named and which on the continent had but recently been identified He went on to say that there had been about 1,000 deaths from it in Danzig in three or four months

From 1865 onwards cases began to be reported in the medical press and from time to time localized outbreaks occurred in widely scattered places Some of these early outbreaks were confused with outbreaks of poliomyelitis which was also on the increase in the early years of the twentieth century The distinguishing features of the two diseases were at this time well known to neurologists but not so well known to doctors in general In 1912 cerebrospinal fever and poliomyelitis became notifiable all over the country, and from 1914 to 1918 there was an epidemic of cerebrospinal fever which was much larger and more widespread than anything which had gone before

This epidemic of the First World War, which began in the winter of 1914–15 was associated with outbreaks in army camps particularly those on Salisbury Plain where the first contingent of Canadian troops was quartered The largest number of cases occurred in the first year (winter 1914–15)

* M Viesseux 1805 † A. Hirsch 1833–6

disease to monkeys and from that time until recently the only method for the detection of most strains of virus was by the inoculation of monkeys Recently Enders* and his colleagues have succeeded in growing poliomyelitis viruses in cultures of living tissues a technique which enormously enlarges the scope of the laboratory investigation of the disease

These viruses have variable effects on different people They can produce anything from a severe and rapidly fatal illness to a carrier state with no symptoms at all and it is generally believed that the paralysis which is the distinctive feature of the disease is a comparatively rare result of infection that the so-called non paralytic cases far outnumber the paralytic and that the healthy carriers outnumber the non paralytic cases There is however some controversy as to the relative proportions of paralytic and non paralytic cases and of healthy carriers which occur in human populations during epidemics Some believe that nearly everyone becomes infected at some time in his or her life whereas others believe that the distribution of the virus is restricted to those in close contact with known cases Why some develop paralysis when they become infected with the virus whereas others develop a mild febrile illness or no illness at all is unknown although some people have acquired immunity from single or repeated exposures to infections which have been insufficient to produce disease In terms of whole populations this factor of silent immunization has been put forward as the explanation for the relative freedom of populations living under primitive conditions from dramatic epidemics of poliomyelitis The theory is that such populations are so to speak, salted and so acquire immunity at an early age

In England and indeed throughout the world authentic records of epidemics of poliomyelitis are historically very recent In England there is a record by Badham† of four cases among children in the small town of Worksop Nottinghamshire in the late summer and autumn of 1834 and after that there is a long interval in which no groups of cases were

* J F Enders 1953 † J Badham 1834

than epidemic there have been about 1 300 cases a year with about 300 deaths but these national figures give a rather gloomy picture of the normal fatality of the disease because the deaths usually occur in the atypical cases where the interval between onset and death is only a few hours Treatment with sulpha drugs and penicillin is effective if started early enough and in some hospitals the case mortality is now very low

The general epidemiological picture of cerebrospinal fever is that of a disease which has been endemic in England for at least a hundred years There have been two epidemic periods associated with the two World Wars and one lesser epidemic period between them The last epidemic produced the largest number of cases, but the disease was less fatal than in previous ones because new and successful methods of treatment had been discovered These have now been improved still more and if a diagnosis is made quickly and treatment is started at once patients usually recover No one knows why epidemics occur but it looks as though one of the most important factors is the sudden aggregation of young adults in overcrowded barracks which occurs at the beginning of a war Possibly the importation of unfamiliar strains of meningococcus by troops from abroad may be important but there is not much evidence of this

ANTERIOR POLIOMYELITIS (INFANTILE PARALYSIS)

The popular name infantile paralysis no longer gives a true picture of the usual age incidence of this disease and so the scientific name anterior poliomyelitis shortened to poliomyelitis or even polio has come so generally into use that it seems best to call the disease poliomyelitis throughout this section

Poliomyelitis is an acute infection due to one of the smallest known viruses or rather to a group of viruses of three main types The presence of a virus was demonstrated by Landsteiner and Popper* in 1908 by transmission of the

* E. Popper 1908

disease to monkeys and from that time until recently the only method for the detection of most strains of virus was by the inoculation of monkeys Recently Enders* and his colleagues have succeeded in growing poliomyelitis viruses in cultures of living tissues a technique which enormously enlarges the scope of the laboratory investigation of the disease.

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certain other countries notably in North America and Scandinavia In the years of high prevalence epidemics were localized in the sense that some counties and cities had dramatic outbreaks but in addition many other places experienced a slight increase in the number of cases There were a few exceptions to this general pattern when only one or two districts had a sharp epidemic in a year while the country as a whole had a year of low incidence In 1947 the total number of cases notified rose suddenly to 7 776 as compared with the previous highest figure of 1,489 in 1938 But the general pattern of distribution in 1947 was not very different in type from that of previous years Once again there were centres of epidemic dissemination with an increased prevalence in many other places The change seems to have been quantitative rather than qualitative Since 1947 the general level of incidence has been much higher than ever before and even the years of low prevalence for example 1948 have outstripped the years of highest prevalence before the Second World War

From the *historical point of view the most interesting* feature about the geographical distribution of poliomyelitis is the tendency for epidemics to recur in certain places sometimes after many years The reasons why an epidemic in area A in one year should extend into the adjacent area B in the next are not difficult to give but why area A should have an epidemic in say 1910 and others in 1926 and 1938 is not easy to explain it is even more difficult to see why when A has had an epidemic in 1910 B the area immediately adjacent should have one perhaps twenty years later And yet this type of thing seems to have happened oftener than can be explained by pure chance The problem has been examined in detail for the period between the wars by Professor Bradford Hill * and the author has added a footnote covering the epidemic wave before the First World War Clearly recurrences in these periods are more impressive as evidence than recent experience because prevalence has been so widespread since 1947 that it would be possible to find instances of recurrence almost anywhere in the

A. Bradford Hill 1947

reported Throughout the nineteenth century single cases *were often shown at medical meetings and knowledge of the* disease gradually became more general but it was only in the last two decades of the century that groups of cases began to be reported in various parts of the world In the late summer of 1891 there was a group of cases of paralysis among children in and near the town of Market Rasen in Lincolnshire This outbreak was described by the medical officer of health in a report which is in the archives of the Local Government Board (now the Ministry of Health) At the time this was thought to be an outbreak of cerebrospinal fever but there are good reasons for believing that seventeen children who fell ill in the period June to October 1891 really had poliomyelitis All of them had some degree of paralysis but only two died This incident is of particular interest because if it was poliomyelitis and not cerebrospinal fever *this is the first authentic epidemic in England In the* nineties there are several reports from neurologists of small groups of cases and in the first decade of the twentieth century there are indications that the disease was on the increase in big cities in 1908 there was an outbreak of eight cases in the village of Upminster Essex followed in 1909 by thirty seven cases in Bristol

In the years immediately preceding the First World War and particularly in 1911 there was an epidemic wave which affected widely scattered areas in the country In 1910 for example the parts chiefly affected were Leicestershire and the *north west coast (Carlisle etc and Barrow in Furness)* whereas in 1911 a widespread prevalence was identified with particularly high incidence in parts of Devon and Cornwall East Anglia Westmorland Birmingham and in the West Riding of Yorkshire During the early years of the First World War little was heard of poliomyelitis but in 1917 the reports of epidemics began again and thereafter up to 1946 the annual reports of the Chief Medical Officer of the Ministry of Health usually included a record of an epidemic somewhere in the country *Between the World Wars two* years 1926 and 1938 were outstanding but even in these the incidence was never on the scale of that experienced in

extensive trials of a vaccine prepared from virus killed by formalin have proved the value of the vaccine The method of control by active immunization is although not giving complete protection the only one which seems likely to be effective because the spread of virus is so often an unseen and therefore uncontrollable affair

Until 1947 England had been fortunate in its experience of epidemic poliomyelitis in comparison with the Scandinavian countries and with North America but since then the attack rates have been much higher than ever before and at present they show no sign of falling to the levels obtaining before 1947

ENCEPHALITIS LETHARGICA

From the historical point of view epidemic encephalitis lethargica has so far been a nine-days wonder Whether it was a disease entirely new to England when it appeared in 1918 is uncertain but the epidemic which began then and petered out about 1930 was certainly unique in authentic historical records After the epidemic period a few cases of encephalitis lethargica were still notified every year but there is considerable doubt whether these were really cases of the same disease or whether they were other forms of encephalitis It is difficult to distinguish between different kinds of encephalitis from signs and symptoms and the causal organism of encephalitis lethargica has never been demonstrated though in other parts of the world several viruses which cause encephalitis have been discovered Encephalitis lethargica is assumed to be due to a virus because of its analogies with other forms of encephalitis due to viruses and because no larger organism has ever been found

The chief symptoms and signs noticed in the early cases seen in London and in Sheffield in March and April 1918 were lethargy with paralysis of cranial nerves particularly of the nerves working the muscles of the eyes but the symptoms of onset were variable One of the most distressing features of the disease was that permanent mental changes

country It is however interesting that some of the places which were affected early in 1947 had been the scene of considerable epidemics in the past

Poliomyelitis is no longer a disease of infants in England or indeed in any of the countries of Western civilization. We have somewhat meagre records of the age distribution of cases before 1944 but there can be no doubt that the proportion of older children and adults attacked in recent years has been much higher than it was in the early epidemics In 1947 about one third of all cases occurred among children under five about one third in children from five to fourteen and about one third in persons aged fifteen and over Children are still more susceptible to the disease, but if adults do contract it they tend to have severe attacks

In all parts of the world poliomyelitis tends to occur in the warmer months of the year but of recent years in England cases seem to have occurred more commonly in the colder months than was previously normal, and there have been instances of definite winter epidemics in other countries

To the student of preventive medicine poliomyelitis appears at first sight to be a thoroughly perverse disease The greatest epidemics have occurred in countries with relatively high standards of living and the disease seems to be just as common and as fatal among members of the higher social classes as among those of the lower It has even been suggested from time to time that members of the higher social classes suffer more though this view rests on impressions rather than on definite evidence The usually accepted explanation for this apparent perversity has already been mentioned it is that the population with relatively low hygienic standards becomes salted whereas that with high standards does not

From time to time attempts have been made to protect individuals against poliomyelitis by passive or active immunization but in the monkey era of laboratory work they were completely unsuccessful Recently the introduction of methods for the culture of virus on living tissues revived interest in the problem of active immunization and

II

MALARIA AND YELLOW FEVER

MALARIA and yellow fever are now generally regarded as tropical or at least hot country diseases but formerly neither disease was confined to the tropics Malaria had a wide distribution in marshy places throughout the world and was at one time quite important in England Yellow fever caused some devastating epidemics in the United States in the eighteenth century of which that in Philadelphia in 1793 is a well known example Though it never became very important in England there was one little outbreak in 1865 which will be described because of its interest as an epidemiological freak

MALARIA

In the world as a whole malaria is still probably the most important communicable disease not so much from the aspect of the number of deaths it causes but because of the amount of chronic ill health which results In England indigenous malaria is now of no importance at all but occasionally a single case of the disease apparently contracted in that country is reported in the medical press as a curiosity

The disease is due to a group of protozoa and the different members of the group produce slightly different symptoms These protozoa have complicated life histories and are carried from the infected person to the uninfected by anopheline mosquitoes Laveran * a French army surgeon discovered the protozoa in the blood of infected patients in 1880 and Ross† demonstrated the truth which had long been suspected that the protozoa were carried by mosquitoes and that they completed a stage of their complicated

* C.A.L. Laveran 1880 † R. Ross 1923

were apt to occur and another permanent effect was a condition resembling *paralysis agitans* In 1918 the disease was reported in fifty one widely separated districts in England and Wales The history of notifications is shown in Figure 16 The year 1924 was the year of highest incidence with just over 5 000 notifications and after that there was a gradual decline

The disease attacked people of all ages and of both sexes and was commoner in the great centres of population than in rural areas The first quarter of the year showed the highest number of notifications with a gradual falling off in the second and third quarters whereas in the fourth incidence began to rise again In most instances there was little evidence of case to case infection but there were striking exceptions to this rule notably an outbreak in a home for girls at Derby in 1919 in which twelve cases with five deaths occurred among twenty two residents There appeared to be no connexion between liability to attack and social conditions

The epidemiological behaviour of encephalitis lethargica and poliomyelitis exhibits many similarities The same wide distribution of cases with occasional dramatic localized outbreaks is typical but the age distribution and the seasonal distribution are quite different The epidemic form of encephalitis lethargica has now disappeared from the records but no one can tell whether the story is ended or is to be continued

Channel coast near Bridgwater Except in the Isle of Sheppey it was generally agreed that the amount of disease had declined very much in the twenty years preceding 1860

By the beginning of the twentieth century indigenous malaria had practically disappeared from England but the two World Wars aroused fears of a possible recrudescence In both large numbers of infected service men arrived back in the country and mosquitoes which could carry the disease were not uncommon There was therefore a theoretical danger that the disease might become endemic in the marshy districts once again

YELLOW FEVER

Yellow fever (Yellow Jack) is caused by a virus carried by certain species of mosquito of which *Aedes aegypti* is the most important An attack of the disease confers a long lasting immunity and in areas where the disease is endemic the native population has considerable herd immunity An effective vaccine is now available for protection against the disease At the present time the disease is confined to the west coast of Africa and to parts of South America but in the seventeenth eighteenth and nineteenth centuries there were many serious epidemics in the ports of the eastern sea board of North America extending as far north as Halifax Nova Scotia (epidemic in 1861) In Europe there were epidemics in Spain and Portugal in the eighteenth and nineteenth centuries but in England though ships have often arrived with cases aboard the disease has spread from a ship to the inhabitants of a port on only one occasion – in Swansea in 1865 This epidemiological curiosity is of sufficient interest to deserve a detailed description The small barque *Hecla* left Cuba on 26 July 1865 On the voyage several of the crew of ten fell ill, and when she arrived at Swansea on 9 September one seaman was dying and two were convalescent The *Hecla* was moored next to a small vessel called the *Eleanor* and in the ensuing weeks twenty inhabitants of Swansea and three other members of the crew of the *Eleanor* developed yellow fever Fifteen of those

life history within the body of the mosquito These fundamental discoveries have been enormously important from the point of view of control in tropical countries, and the applied science of malariology has grown up and is concerned with the peculiarities of the different parasites the ecology of the various anopheline mosquitoes and the devising of methods for their destruction From the parochial point of view it is however salutary to remember that these fundamental discoveries had nothing to do with the disappearance of indigenous malaria from England It had gone before the discoveries were made as a result of social change which was not directed specifically to the control of malaria

The early history of malaria in England is lost in a confusion of names Creighton* points out that originally the word ague, which later became the popular name for malaria did not mean a paroxysmal or intermittent fever but merely an acute one Sydenham† frequently uses the term ague to describe various epidemics of febrile diseases but he makes only one reference to marsh agues This single reference is in his essay on hysteria where he interpolates the remark that the blood of a person who has spent two or three days in a locality of marshes and lakes is in the first instance impressed with a certain spirituous miasma which produces quartan ague It is clear that Sydenham and his contemporaries knew of the endemic ague of marshy districts, but did not regard it as of much importance in comparison with all the other acute fevers which came in epidemics all over England

We have to wait until Simon s‡ tenure of office under the Privy Council for any serious attempt to estimate the prevalence of malaria in England In Simon s sixth report (p 430) there is a section by Dr G Whitley on the prevalence of ague and other malarious conditions in the marshy districts of England He found that the Isle of Sheppey was the chief focus of the disease, but that there was still some in the Fen country and in *the marshy districts of the Bristol*

* C. Creighton 1894. † T Sydenham 1676 ‡ John Simon 1860

12

TUBERCULOSIS

TUBERCULOSIS is different from the diseases considered so far in that it has never been epidemic in Britain in the sense of being much more prevalent at one time than at another It has however been an important cause of death throughout the period covered by this book and probably for much longer

The disease is caused by *Mycobacterium tuberculosis* discovered by Robert Koch* (1843–1910) in 1882 and two types the human and the bovine cause disease in man Many different organs can be affected but disease of the lungs is by far the most common form and the easiest to trace in historical records Laennec (1781–1826) the French physician who invented the stethoscope recognized the essential unity of tuberculosis of different organs but the final proof of this unity did not come until Koch s discovery of the tubercle bacillus and so earlier statistical records of forms of tuberculosis other than respiratory are particularly suspect because of difficulties of classification and confusion with other diseases In considering the early history of tuberculosis it is better to confine the discussion to disease of the lungs than to attempt a review of all forms of the disease

PULMONARY TUBERCULOSIS

EARLY HISTORY

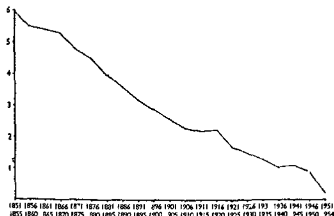
The earliest medical books by English physicians about pulmonary tuberculosis (consumption phthisis) were written in the seventeenth century In this century Christopher Bennett (1617–55) Thomas Willis (1621–75) Thomas Sydenham (1624–89), and Richard Morton (1639–98) all

* R Koch 1882

attacked died The three members of the crew of the *Eleanor* actually developed the disease at Llanelly The weather at this time was extremely hot with temperatures of 80 to 90 F in the sun and 70 to 80° in the shade

This unique experience has a little relevance to the present day since the aeroplane has brought the tropics to our door step and it is always a possibility that a person in the in cubation stage of yellow fever may arrive and develop the disease in England We must remember however that the Swansea incident is unique and happened long before the part played by mosquitoes in carrying the disease was known Nowadays strict precautions are taken against the importa tion of infected mosquitoes and there are also strict rules about the vaccination of travellers Perhaps the greatest safeguard is the fact that England seems to be a most un favourable soil for the propagation of epidemics of yellow fever In the days before much was known about the disease there must have been many undetected importations of Yellow Jack particularly from the West Indies and yet so far as we know the Swansea outbreak is the only occasion on which the disease spread to the native population and that to a very limited extent

causes The crude death rate for the quinquennial period 1838–42 was 3 880 per million There is a gap in the statistical record in the forties because the number of deaths by cause was not abstracted for the years 1843–6 The figures for the years 1851–5 suggest however that there was a slow fall in the death rate in the forties In this quinquennium (1851–5) the total number of deaths averaged about 400 000 a year and those from tuberculosis of the lungs amounted to about 51 000 a year The crude death rate for the years



17 *Comparative mortality index showing trend of respiratory tuberculosis in England and Wales 1851–1954 (1938=1)*

1851–5 was 2 851 per million. The trend of mortality from 1851 to 1954 is shown in Figure 17 This graph is derived from table six of the Registrar General's *Statistical Review* (Part 1) 1954 In this table he gives comparative mortality indexes for quinquennial periods from 1851–5 to 1931–5 for the four year period 1936–9 immediately preceding the outbreak of the Second World War and subsequently for the quinquennial periods 1940–4 to 1950–4 The base year for all comparative mortality indexes is the year 1938 which is taken as unity Thus mortality in 1851–5 was nearly six times what it was in 1938 whereas in 1950–4 it was 0 25

either wrote whole books about the disease or devoted chapters to it in general text books Morton s book, *Phthisiologia* published in 1688 was the most comprehensive and important of these contributions Much of the theoretical matter in these books is now out of date but the descriptions of cases make it clear that the authors were in the main writing about tuberculosis of the lungs as we know it today that the disease was common and that it was an important cause of death The Bills of Mortality confirm this, for in most years between fifteen and twenty per cent of all deaths in London were returned under the heading consumption In plague years particularly in 1665 the proportion was much lower because the deaths from plague were so many

Brownlee* reviewed the evidence of the Bills for the whole period 1629–1838 during which causes of death were distinguished He came to the conclusion that there was an increase in the death rate from pulmonary tuberculosis in London in the eighteenth century which culminated about 1800 and that there was a slow fall in mortality from then on For the statistical period (1838 onwards) this fall is well authenticated but the statistics of the earlier period are too doubtful to make it more than a probability For the country as a whole there are no statistics before 1838 but the growth of towns through immigration from the country districts was going on rapidly and these new aggregations of susceptible people must have been favourable soil for the spread of the disease The probability is therefore that the rise in mortality which affected London in the eighteenth century affected the industrial towns even more and probably reached its zenith rather later in them than in London

FROM 1838 TO THE PRESENT DAY

The trend of mortality at all ages

In the first five complete years of registration the number of deaths assigned to tuberculosis of the lungs was about 60 000 a year out of a total of some 350 000 deaths from all

* J Brownlee 1916

times that of 1938 The advantage of using the comparative mortality index (C.M I) rather than the crude death rate for a long term study is that the C M I makes allowance for the great changes in the age distribution of the popula tion which have taken place in the last hundred years

The recent trend of annual mortality at all ages is shown in Figure 19 which overlaps the long term graph (Figure 18) The effect of the two World Wars is shown more clearly than in the long term graph and the prolongation to 1955 brings out the rapidity of the decline of mortality since 1947 The index fell from 1 8 to 1 0 in the seventeen years 1921–38 and from 0 93 to 0 25 in the eight years 1947–55 In this last eight years the new drugs streptomycin para amino-salicylic acid (P A S) and isoniazid (I N H) have come into general use and there have been important advances in surgical treatment

Incidence

Figure 19 serves as a link between the discussion of mortality and that of incidence The lower curve is virtually the same as the second half of that shown in Figure 18 but in constructing it the absolute numbers of deaths each year have been plotted instead of the comparative mortality indexes The total deaths fell from about 27 000 in 1932 to 20 000 in 1947 and 5 837 in 1955 Notifications on the other hand have hardly fallen at all It may be that im proved methods of ascertainment particularly by mass radiography have had some effect in keeping up the num ber of notifications but even allowing for this possibility it seems probable that there has been no decline of incidence comparable to that of mortality

Changes in the age and sex distribution of mortality

Although mortality at all ages has been falling through out the statistical period there have been differences in the rate of decline in different age groups For example the rate of mortality among young women which has always been higher than that of young men fell very little between about 1900 and 1932 Recently however the death rates



18 *Comparative mortality index for respiratory tuberculosis in England and Wales 1905–55*

50 000
40 000
30 000
20 000
10 000
Notifications
Deaths
1932
1935
1940
1945
1950
1955

19 *Respiratory tuberculosis Notifications and deaths in England and Wales 1932–55*

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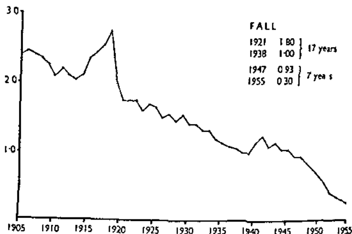
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of young men and young women have been falling very quickly much more rapidly than those of middle aged people and there has also been a lessening of the difference between young men and young women Modern methods of treatment are highly successful in young adults but less successful in the middle aged

Social Conditions including occupation

The Registrar General s studies of occupational mortality have always shown in tuberculosis a rising gradient from social class I (the professional classes) to social class V (unskilled workers) In 1950 according to the analysis of the one per cent sample of the population the standardized *mortality of men in social class V was 2 3 times that of men in social class I*

Within the sub-classes mortality was high among members of the armed forces dock labourers and hewers and getters of coal It was particularly low in agricultural workers The peculiar dangers of dust containing silicon have long been known, and in the supplement to the thirty fifth report of the Registrar General (years 1861–70) Farr drew attention to the fact that steel grinders ran a much greater risk from dust than did those breathing vegetable dusts such as millers The recognition of this risk has led to a whole series of Acts of Parliament and regulations designed to protect workers in dusty industries and to ensure compensation for those who do contract disease when working in a dusty atmosphere Though dusts do not themselves cause tuberculosis they may make the lungs more vulnerable to the effects of infection by *tubercle bacillus*

PREVENTION OF TUBERCULOSIS

There are three lines of attack in the prevention of any infectious disease namely

1 Stamp out the reservoir of infection
2 Discourage the vector
3 Protect the individual

With tuberculosis all three methods are of value to us and although it is not yet possible to win complete success the amount of success which can be achieved depends on how well they are applied

The reservoir of infection in tuberculosis is the so-called open case the patient who has tuberculosis of the lungs and is coughing up live tubercle bacillus This reservoir may be further considered under two headings

(a) The known reservoir of infectors i e the known cases of open pulmonary tuberculosis and treatment at clinics and hospitals in England and Wales the number of these cases in 1955 was estimated to be approximately 20 000
(b) The unknown reservoir of infectors i e unknown cases of open pulmonary tuberculosis still at large amongst the general public It is difficult to give an exact figure for this but if mass radiography experience is representative of the whole population there are about 75 000 unsuspected cases of respiratory tuberculosis in the community at any one time or one in every 600 of the population

Where the patient is known and attending regularly for treatment, much can be done to prevent the spread of the disease It is not always possible even with modern drugs to render him not infectious but much can be done to minimize the risk by instructing him in methods of personal hygiene and disposal of sputum

The unknown infector patient is much more dangerous and the finding of such patients is perhaps the most important task in the preventive service of today Mass radiography surveys have proved of great value in this respect but there remains a small group of elderly bronchitics whom it is very difficult to persuade to attend

Our last line of attack against tuberculosis protecting the individual has been much used in this country in recent years It consists of vaccinating the healthy person with a live but attenuated strain of tubercle bacillus known as the Bacille Calmette Guerin (B C G)

This vaccine was brought into experimental use as long ago as 1924 but received a severe setback in 1930 in the Lubeck disaster where, owing to a mistake in culture children were vaccinated by an active virulent bacillus and 73 died of tuberculosis

The vaccine is now manufactured in an isolated laboratory completely separated from any other building and having its own staff

The vaccine gives good protection for some five to seven years In England it is used to protect all children who have to live in a tuberculous household all nurses and staff of tuberculosis clinics and hospitals and lastly it is given to school children of fourteen years of age to afford protection in these adolescent years as it is in the late teens when these children first go out into the world that tuberculosis is most likely to develop

13

THE RELATIVE IMPORTANCE OF THE DIFFERENT DISEASES AT DIFFERENT TIMES

The characters in this book are diseases and in order that we may see them in relation to one another at different periods of history it is interesting to analyse the deaths by cause in certain years when one or other disease has been at its zenith This is a rough and ready method of comparison which introduces a little more precision into the story than the bare *magna mortalitas* of the chroniclers of the Black Death As we progress through the centuries the picture becomes clearer and the year 1838 when the Bills of Mortality were replaced by proper registration is a most important landmark From then on the figures are far more accurate and comprehensive than they had ever been before

1665

The year of the Great Plague is the most convenient starting place because it marks the end of that period lasting nearly 300 years during which plague dominated the scene so much that other epidemic diseases paled into insignificance beside it The complete Bill for 1665 (see Appendix p 149) gives a total of 97 306 burials whereof of the plague 68 596 The heading which comes next numerically is that called ague and feaver with 5 257 burials It seems likely that some of the deaths really due to plague were returned under this heading since the number is considerably swollen in comparison with the figures of other years In 1664 for example there were 17 453 burials in the Bills and 2 278 of them were ascribed to ague and feaver In the whole period of five years 1660–4 there were some 71 000 burials noted in the Bills – only about 2 000 more than the number

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in young children – rather than to an increase in the birth rate Edmonds * writing in the *Lancet* of 1835–6 made some simple calculations from the Bills of Mortality which show that there was a substantial fall in the mortality of young children in London and it seems reasonable to suppose that the fall was even greater in the far healthier countryside

	1730–49	1750–69	1770–89	1790–1809	1810–29
Annual baptisms (average)	15 773	15 370	17.474	19 320	23 896
Annual deaths of children under 5 years old (average)	11 754	9 685	9 003	7 979	7 590
Deaths per cent of baptisms	75	63	52	41	32

The rise in the number of baptisms may seem inconsistent with the statement that there was no great increase in the birth rate in this period but it must be remembered that London differed from the country as a whole in that it grew not only by natural increase but by immigration from the rest of the country

From the strictly medical point of view it is not easy to see exactly why the decline of the death rate began when it did There were no great discoveries in the fields of preven tion or of the treatment of diseases There was however a little lightening of the shadows Throughout the second half of the eighteenth century hospitals and dispensaries for the sick poor were being founded by charitable subscrip- tions both in London and in the industrial areas which were growing so rapidly Distinguished physicians like Lettsom working at the General Dispensary in Aldersgate Street Clerk of Newcastle Ferriar of Manchester and Currie of Liverpool were beginning to penetrate into the slums and to write about the horrors which they found there In par ticular they wrote about the prevalence of epidemic typhus which was by this time almost unknown among the more prosperous citizens Social historians have laid stress on the

* T R. Edmonds, 1835.

ascribed to plague alone in 1665 Never since then has the mortality from an epidemic disease been so overwhelming Small wonder that a disease which could cause as many deaths in a year as usually occurred in five years from all causes was an ever present shadow over the lives of our ancestors particularly those who lived in London!

1772–1814

The year 1772 is of particular interest because it was the year in which deaths from smallpox in the London Bills reached their zenith of 3 992 (see Figure 7a) The position of smallpox was however nothing like that of plague in the seventeenth century because deaths from fever' probably chiefly typhus fever were not far behind being 3 207 in 1772 The total number of deaths in the year was 26 053 so that smallpox and fever between them accounted for 28 per cent of the total The nomenclature of diseases in the Bills was in some respects archaic even for the eighteenth century, and this makes it impossible to describe the trend of many diseases The number of deaths from both smallpox and fever almost certainly declined a little in the last thirty years and those from smallpox declined rapidly in the early part of the nineteenth century (see Figure 7b) On the other hand deaths attributed to measles and whooping cough increased though it is impossible to be sure whether this was a real increase or due to rather better reporting

The whole period of the second half of the eighteenth century is of great general interest because it marked the beginning of the rapid growth of population which lasted throughout the nineteenth century and only began to slow down at the end of the first decade of the twentieth There was no census until 1801 and so earlier statistics of population are to some extent controversial but between 1750 and 1801 (first census) the population of England and Wales probably increased from about 6½ millions to about 9 millions and that of London from about 675 000 to about a million It is generally accepted that the increase was due to a decline in the death rate – particularly the death rate

In the first half of the nineteenth century there were two main trends influencing social conditions the one favourable to the public health and the other unfavourable and the two cancelled one another The favourable factors were chiefly better feeding and more enlightened medicine the unfavourable factor the enormous and uncontrolled growth of the great industrial towns Social conditions in these new towns tended to be worse than those in London since many had no form of local government capable of ensuring even the most elementary standards of public hygiene

So far as individual diseases were concerned the number of smallpox deaths declined considerably in London (see Figure 7b) and probably elsewhere Deaths from fever declined in London and probably increased in the large ports and industrial towns particularly in Liverpool which received many Irish immigrants Typhus was probably the most important fever but there can be no doubt that there was typhoid too and in August 1826 Hewett (quoted by Creighton*) of St George s Hospital published an account of ten fatal cases of a fever which was almost certainly typhoid The common infectious diseases of childhood began to occupy an important place in the London Bills of Mortality and in the writings of medical men in other cities (cf Watt† in Glasgow)

In 1831 there began the first epidemic of Asiatic cholera important enough in itself but even more important in its results because it was the fear of cholera which made people realize how bad sanitary conditions were and so led to the series of Public Health Acts of which that of 1848 was the first.

The events which led up to the Act of 1848 are not strictly relevant to the history of diseases but mention must be made of the establishment of the General Register Office in 1837 and of Chadwick s‡ *General Report on the Sanitary Condition of the Labouring Population of Great Britain* of 1842 The General Register Office collected the facts about life and death for the whole country and Chadwick s report drove

* Creighton 1894 † R Watt 1813
‡ Sir E Chadwick 1837

contribution which the doctors made to the reduction of the death rate but general social causes must have been important too Throughout the eighteenth century methods of farming were improving rapidly and the reforming land lords were producing food for the growing populations of the towns on land which had hitherto produced enough for the local inhabitants only The quality of food was improving because root crops made it possible for farmers to keep their animals throughout the winter instead of killing most of them in the autumn These fundamental changes must have improved the diet of the poor and any improvement would reduce the general mortality in particular that of typhus which is pre eminently a disease of famine By modern standards the diet of the poor of the cities was still appalling but we have to compare it with what went before and not with what came after Although the period of sanitary reform came considerably later in the second half of the nineteenth century, there were beginnings towards the end of the eighteenth The sheer discomfort of living in cities with no proper drainage, water supply, or refuse disposal made elementary improvements essential but conscious efforts to improve the public health by sanitary measures were long delayed For example, up to 1815 it was illegal to connect the drains from a house to a public sewer and the houses of the rich had cesspools under the basement Sanitation in the poorer quarters of cities was unspeakably crude Once again however we must make comparisons with what went before and say that there was a little improvement

1815–48

After 1815 the population continued to grow at a rapid rate but the rate of growth was already established and there does not appear to have been any outstanding alteration in the birth or death rates Once the death rate has fallen below the birth rate to any considerable degree a population will continue to grow without any further alteration of rates or even in spite of a degree of adverse change *

* M C Buer 1926

stables the growth of cities had created but three names stand out Edwin Chadwick (1800–90) was the ruthless administrator William Farr the reflective student and research worker while John Simon (1816–1904) comes somewhere between the two Chadwick s official life from his appointment as an Assistant Commissioner of the Poor Law in 1832 to his compulsory retirement from the General Board of Health in 1854 was relatively short, but his achievement was great and his contribution to the future of public health incalculable Farr was appointed compiler of abstracts to the General Register Office in 1839 and retired in 1879 Thus he was largely responsible for the analysis of the information about life and death in this country for the first forty years of general registration He supplied the ammunition which Chadwick and Simon used in their war against dirt and disease Simon was appointed Medical Officer of Health for the City of London in 1847 and from 1855 to 1876 served the various government departments which were successively charged with the care of the public health Simon s period of office at the Privy Council (1858–71) was his golden age In spite of the fact that the legal powers of the Privy Council were limited Simon promoted a number of inquiries into the incidence and mortality of diseases in different districts which had an enormous influence in promoting sanitary reform

In the supplement to the thirty fifth *Annual Report* of the Registrar General (1861–70) Farr traced the march of an English generation through life and compared the mortality experience of the healthy districts with that of England as a whole and with that of Liverpool which was then the supreme example of an unhealthy district His table showing the numbers of children dying under five years of age from various causes is an admirable summary of the relative importance of the principal epidemic diseases in the years 1861–70 The part of Farr s table which relates to epidemic diseases is reproduced on p 138

Appalling as the facts revealed in this table were they were not a complete picture There was another indefinite heading in the table – Brain Diseases – which included a

them home to many influential people who had little idea of how the other half of the world lived

1848–75

The year immediately following the first Public Health Act is an interesting one in which to take another look at the prevailing epidemic diseases of London because cholera is shown at its worst It must be remembered that the Registration Area was much larger than that of the Bills and that the information collected though still defective, was much more complete The Registration Area had a population of 2 360 000 and 68 755 deaths were registered in the year Of these deaths 26 243 were ascribed to the seven principal epidemic diseases

Cholera	14 125
Diarrhoea	3 463 (chiefly deaths of infants)
Typhus	2 482 (typhoid included with typhus)
Whooping cough	2,349
Scarlatina	2 149 (including diphtheria*)
Measles	1,154
Smallpox	521
	26 243

Some of these figures were understated as deaths of infants resulting from epidemic diseases were often returned under indefinite causes such as convulsions The number of births was 72 612 and of deaths among children under five years of age 24 999 so that if we express the number of deaths under five as a percentage of the births as Edmonds† did for earlier years (see table on p 133) we arrive at the figure 34 – not very different from the figure for the years 1810–29

The period between the first Public Health Act of 1848 and the great consolidating Act of 1875 was one of intense activity in the field of preventive medicine Many men played important parts in the cleansing of the Augean

* It is doubtful whether diphtheria was of any importance at th s time

† T R Edmonds 1835

stables the growth of cities had created but three names stand out. Edwin Chadwick (1800–90) was the ruthless administrator William Farr the reflective student and re search worker while John Simon (1816–1904) comes some where between the two Chadwick s official life from his appointment as an Assistant Commissioner of the Poor Law in 1832 to his compulsory retirement from the General Board of Health in 1854 was relatively short but his achievement was great and his contribution to the future of public health incalculable Farr was appointed compiler of abstracts to the General Register Office in 1839 and retired in 1879 Thus he was largely responsible for the analysis of the information about life and death in this country for the first forty years of general registration He supplied the ammunition which Chadwick and Simon used in their war against dirt and disease Simon was appointed *Medical Officer of Health for the City of London in 1847* and from 1855 to 1876 served the various government de partments which were successively charged with the care of the public health Simon s period of office at the Privy Council (1858–72) was his golden age In spite of the fact that the legal powers of the Privy Council were limited Simon promoted a number of inquiries into the incidence and mortality of diseases in different districts which had an enormous influence in promoting sanitary reform

In the supplement to the thirty fifth *Annual Report* of the Registrar General (1861–70) Farr traced the march of an English generation through life and compared the mortality experience of the healthy districts with that of England as a whole and with that of Liverpool which was then the supreme example of an unhealthy district His table show ing the numbers of children dying under five years of age from various causes is an admirable summary of the relative importance of the principal epidemic diseases in the years 1861–70 The part of Farr s table which relates to epidemic diseases is reproduced on p 138

Appalling as the facts revealed in this table were they were not a complete picture There was another indefinite heading in the table – Brain Diseases – which included a

them home to many influential people who had little idea of how the other half of the world lived

1848–75

The year immediately following the first Public Health Act is an interesting one in which to take another look at the prevailing epidemic diseases of London because cholera is shown at its worst It must be remembered that the Registration Area was much larger than that of the Bills and that the information collected though still defective, was much more complete The Registration Area had a population of 2,360 000 and 68 755 deaths were registered in the year Of these deaths 26 243 were ascribed to the seven principal epidemic diseases

Cholera	14 125
Diarrhoea	3 463 (chiefly deaths of infants)
Typhus	2 482 (typhoid included with typhus)
Whooping cough	2 349
Scarlatina	2,149 (including diphtheria*)
Measles	1 154
Smallpox	521
	26 243

Some of these figures were understated as deaths of infants resulting from epidemic diseases were often returned under indefinite causes such as convulsions The number of births was 72 612 and of deaths among children under five years of age 24 999 so that if we express the number of deaths under five as a percentage of the births as Edmonds† did for earlier years (see table on p 133) we arrive at the figure 34 – not very different from the figure for the years 1810–29

The period between the first Public Health Act of 1848 and the great consolidating Act of 1875 was one of intense activity in the field of preventive medicine Many men played important parts in the cleansing of the Augean

* It is doubtful whether diphtheria was of any importance at this time
† T R Edmonds 1835

End of London where the new sewage system was not yet completed The last great outbreak of classical smallpox occurred in 1871–2 and in the two years there were 42 000 deaths in England and Wales nearly 10 000 of them in London

1875–1900

In 1889–92 there was a pandemic of influenza which affected England chiefly in 1891–2 Ever since then influenza has been an important epidemic disease

After 1872 the death rate of smallpox declined rapidly Scarlet fever was the most fatal of the common infectious diseases of childhood at the beginning of the period but at the end of the century measles and whooping-cough came almost equal first, diphtheria second and scarlet fever last with a death rate less than half that of measles or of whooping-cough

The death rate and probably the incidence of the enteric fevers (typhoid and paratyphoid) declined between 1871 and 1890 and then remained constant until 1900 The decline was probably associated with the improvements which were being made in public water supplies These improvements in the public health affected the mortality of adults and of older children more than they affected that of babies and infant mortality in the quinquennium 1895–1900 was even a little higher than in the decennium 1841–50

In this last quarter of the nineteenth century the new science of bacteriology came into its own and discoveries of micro-organisms which cause disease were announced nearly every year At first the discovery of the micro-organism which caused a disease seemed likely to lead to the effective control of that disease but soon it became evident that control was not so easy The discovery of the existence of healthy carriers or organisms which could produce disease in others made it obvious that with some diseases the isolation of those who had obvious symptoms only would never prevent spread

Studies of immunity which had their origin in Pasteur s work on chicken cholera anthrax and rabies were also

large number of deaths of young children from one of the acute infectious diseases In the Liverpool district there were 49 840 deaths under this heading Thus in Liverpool nearly half the children born died before the age of five, and even in the healthy districts just over one sixth died before that

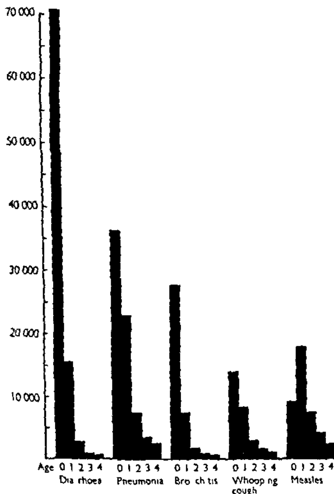
Table showing the number of deaths of children under five out of 1 000 000 children born alive (1) in the healthy districts (2) in all England (3) in the Liverpool district (Based on the returns for the years 1861–70)

	Healthy Districts	England	Liverpool
Deaths from all causes	175 410	263 182	460 370
Smallpox	602	3 331	5 175
Measles	5 257	11 507	25 514
Scarlatina	11 373	17 959	26 818
Diphtheria	4 184	2,425	3 395
Whooping-cough	9 650	14 424	32 551
Typhus (with enteric and common fever)	2 807	5,401	9 297
Diarrhoea and dysentery	9 354	20 344	51 911
Cholera	399	1 129	4 255
Other zymotic diseases	6 135	10 579	12 093
Total zymotic diseases	49 761	87 099	171 009

(N B Zymotic diseases was the term roughly equivalent to the modern term Acute Infectious Diseases)

age Farr does not give a detailed table for London but he gives a summary which shows that the figure for deaths from all causes was 304 518 Thus London came between Liverpool and the whole of England

Figures for the decennial period provide a good idea of the general level of mortality from epidemic diseases but they do not show how disastrous were the effects of individual epidemics In 1863 and 1873 for example more than 30 000 deaths were ascribed to scarlet fever and in London alone there were about 6 000 deaths from this disease in 1869 and in 1870 The last epidemic of cholera was in 1865–6 It was smaller than its predecessors and affected chiefly the East



20 *Deaths from diarrhoea pneumonia bronchitis whooping-cough and measles of children under five in England and Wales 1911–14 (age 0 = infants from birth to first birthday)*

going on in this period and in 1890 von Behring* (1854-1917) made an antitoxin which was effective in the treatment of diphtheria

1900–1914

The decline in the death rate of diphtheria which began in the early part of the twentieth century was probably due largely to the introduction of antitoxin (see Figure 13) The death rate of the enteric fevers which had remained constant from about 1885 to 1900 also began to fall about 1900 and it seems plausible to suppose that this second fall was associated with the greater precision which bacteriology brought to measures of control (see Figure 8)

The greatest change in the trend of mortality in the early twentieth century related however to infants The infant mortality rate had been almost constant at about 150 deaths per thousand live births from the beginning of registration (1838) to 1901 but since then it has declined steadily except for small setbacks in the two World Wars and in 1954 the rate was 25 4 There are many indefinite causes of death in infancy and it is therefore difficult to make valid generalizations about the causes of this decline but general improvements in social conditions and better care of infants seem to have been more important than advances in methods of treatment The reduction in the size of families may have been important because a school child often brings infectious disease into its home and many infections are more fatal to babies than they are to older children

In the years immediately preceding the outbreak of the First World War the epidemic diseases which had been important causes of death among older children and adults had become relatively unimportant Typhus cholera and smallpox had virtually disappeared the enteric fevers were much less common and scarlet fever though probably as prevalent as ever, was less fatal Influenza was at this time more a nuisance than a serious cause of death Although the death rate of diphtheria had declined slowly, it was the

* E von Behring and Wernicke 1892

reported in the years 1911–13 In this period the disease chiefly attacked children under five and so its popular name of infantile paralysis was an appropriate one As a cause of death it was insignificant in comparison with the common infectious diseases and it was not even mentioned in the report from which the table on p 142 and Figure 20 were taken As a cause of crippling poliomyelitis has been important ever since 1910 and it became relatively more prominent as tuberculosis of bones and joints declined

1914–18

In the winter of 1914–15 cerebrospinal fever appeared in certain army camps particularly in those on Salisbury Plain where the first contingent of Canadian troops was quartered In the First World War the disease affected the services more than civilians and outbreaks were commoner among recruits than among seasoned troops The outstanding epidemic of the war years was however that of influenza which began in the early summer of 1918 and reached a climax in the autumn and winter An exact comparison with the great epidemics of earlier years is impossible but anyone who lived through it must remember that nearly everyone had the disease The number of deaths attributed to influenza in the two years 1918–19 was 157 000 but there is no doubt that many deaths really due to influenza were returned under other headings particularly that of pneumonia The deaths ascribed to pneumonia were much higher in these two years than in those preceding or following them Influenza killed at least three times as many people in England and Wales as did the worst epidemic of cholera in 1849 but between 1849 and 1918 the population had increased from about 17 millions to 37 millions The chief feature of the pandemic of 1918–19 was that all over the world the disease was more fatal to those in the prime of life than it had ever been before or has been since

In March and April 1918 the first cases of encephalitis lethargica were reported in London and in Sheffield There had been an epidemic of the disease in Vienna in the war years but doctors in England only heard about it after the

most important of the common infectious diseases as a cause of death in children of school age

The epidemic diseases which affected young children particularly were still a serious public health problem and the Local Government Board prepared a series of reports on infant and child mortality in order to emphasize the need for improvements in the child health services One of these dealt with the causes of death among children under five in England and Wales in the four years 1911–14 In this age group there were four diseases or groups of diseases which were major causes of death bronchitis and pneumonia, summer diarrhoea, measles whooping cough There were important differences in mortality in different types of area and in the relative importance of the different diseases in different age groups The following table illustrates the first point and Figure 20 the second

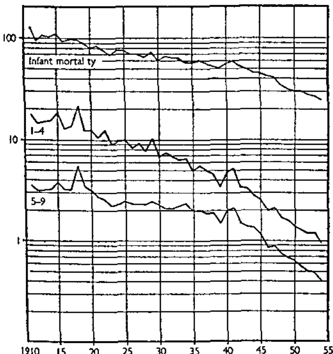
	England and Wales	Large towns	Smaller towns	Other towns	Rural districts	London
Infant mortality	110	122	113	104	90	108
Deaths of children under 5 per 1 000 births						
All causes	164	188	167	152	125	164
Measles	*12*	*15*	*12*	*10*	*6*	16
Whooping-cough	8	9	8	8	7	8
Bronchitis and pneumonia	32	38	32	29	23	33
Diarrhoea	26	32	28	21	15	30

The figures in the London column may be compared with those of Edmonds in the table on page 133

Nearly half the deaths ascribed to summer diarrhoea occurred in one season of one of the four years the exceptionally hot summer of 1911 and so the above table gives an exaggerated impression of the general importance of summer diarrhoea The authors of the report could not know what the future trend of summer diarrhoea would be but in fact the last great epidemic was that of 1911 (see Figure 10)

The earliest widespread epidemics of poliomyelitis were

two diseases did not change either and that the decline of their death rates was entirely due to the fact that they became less fatal Deaths from measles and whooping cough are usually caused by complications rather than by the



21 *De th ales (all causes) of h ld en in d ff rent g groups in E land and W al s 1911–55 (on logarithmic paper to facil tate comp risons b twe n the rates of decl ne in the d fferent a e g oups)*

end of the war and were at first puzzled by the bizarre symptoms and signs

1919–39

The influenza epidemic of 1891–2 was followed by a prolonged rise in the general level of mortality from the disease, and in the years following that of 1918–19 there was a further step-up The highest rate of the years between the World Wars was recorded in 1929 and after that there was a slow decline of the death rate both in epidemic and in non epidemic years (see Figure 6)

The death rate from the enteric fevers maintained the steady decline which had begun in 1900 Incidence, how ever after a steep fall between 1911 and 1920 showed no consistent trend in the twenties In the thirties the general level of incidence was lower but was high in 1936 and 1937 chiefly because of the epidemics of typhoid fever in Bourne mouth (1936) and Croydon (1937)

Summer diarrhoea of infants had almost disappeared by 1920 but there was a small rise in the infant mortality from this cause in the hot summer of 1921

There were interesting differences between the trends of mortality from the common infectious diseases of childhood At the beginning of the period the death rates of diphtheria measles and whooping cough among children under fifteen were not very different from one another That of scarlet fever was much lower By 1939 mortality from measles and whooping cough had declined so much that diphtheria had become easily the most important of the common infectious diseases particularly among children of school age Mortal ity from scarlet fever had declined still further and by 1939 the disease was of no importance as a cause of death From 1911 onwards national statistics of notifications of diphtheria and scarlet fever were collected and these showed that there was no long term change in the trend of incidence of the two diseases Measles and whooping cough were not made notifiable until 1940 and so there are no national statistics of their incidence between the World Wars It seems probable, however, that the long term trend of incidence of these

children was temporarily reversed The epidemic of cerebro-spinal fever unlike that of the First World War affected the civilian population more than the services The disease was less fatal than it had been because the sulpha drugs were far more effective than any treatment which had been available before. In 1941 the comprehensive scheme for the immunization of children against diphtheria was inaugurated and in the years since then the disease has almost disappeared

The later war years were uneventful and the decline of the death rates of infants and children was even more rapid than it had been before the beginning of the war

In 1946 there were no fewer than fifteen importations of severe smallpox and in this and the following year there was an increase in the number of cases to 56 in 1946 and 78 in 1947

The most important event of 1947 was the sudden and unexplained increase in the prevalence of poliomyelitis At the end of June it became clear that the usual seasonal rise in notifications was much greater than in any previous year and in the week ended 5 July more notifications were received than there had been in the highest week of 1938 The peak of the epidemic came in the week ended 6 September when 708 cases were notified compared with the previous record of 85 cases in a week in October 1938 In the whole year there were about four and a half times as many cases as in 1938 Since the end of June 1947 poliomyelitis had been the most prominent epidemic disease of England There have been good and bad years but the general level of incidence has been higher than it ever was before and so far there is no sign of a consistent downward trend

Since 1948 the Chief Medical Officer of the Ministry of Health has included in his annual report a short section on the noteworthy outbreaks of epidemic diseases in each year A rough analysis of these is of interest in that it gives a general picture of the epidemic situation at the present time In the period there have been two small outbreaks of typhoid fever and three of paratyphoid Outbreaks of food

different age groups of childhood from 1911 to 1955 The most prominent peaks on the curves of age groups 1–4 and 5–9 are in 1918 and were due to influenza The lesser peaks are most obvious in the curve for age group 1–4 and were all associated with high prevalence of one or more of the acute infections For example, that of 1922 was due to high prevalence of whooping-cough and of measles and that of 1929 to epidemics of whooping cough and influenza

In the last years of the war cerebrospinal fever declined and throughout the twenties incidence and mortality remained fairly constant In the early thirties there was a further period of activity with a peak in the years 1931–2 There was no consistent decline in fatality like that of measles and whooping cough The prevalence of encephalitis lethargica rose to a peak in 1924 and then gradually declined Poliomyelitis occurred throughout the period but the outbreaks were usually scattered and the incidence was never on the same scale as that experienced in the same period in the Scandinavian countries and in North America There were two years of particularly high incidence – 1926 and 1938 – and incidence in 1938 was higher than that in 1926 This disease was not important as a cause of death, but as other crippling diseases declined it became an important cause of permanent paralysis of limbs There are no comprehensive statistics of age incidence for the inter war years, but the figures for London suggest that after about 1930 there was a rise in the average age of attack.

The chief trends in the inter war years were then, a further decline in the incidence of the enteric fevers a great decline in the fatality of measles whooping cough and scarlet fever, little change in the incidence or severity of cerebrospinal fever and poliomyelitis

THE SECOND WORLD WAR AND AFTER

In the years 1940 and 1941 there was a sharp rise in the notifications of paratyphoid fever and a small rise in the death rate from this disease In the same years cerebrospinal fever had the highest recorded prevalence since that of 1915 and the favourable trend of death rates of infants and

A general Bill for this present year, ending the 19 of *December* 1665 according to the Report made to the KINGS most Excellent Majesty

By the Company of Parish Clerks of *London* &c.

	Buried	*Pla*		*Buried*	*Pla*
St Albans Woodstreet	200	121	St James Dukes place	262	190
St Alhallowes Barking	514	330	St James Garlickhithe	189	118
St Alhallowes Breadst	35	16	St John Baptist	138	83
St Alhallowes Great	455	426	St John Euangelist	9	–
St Alhallowes Honila	10	5	St John Zacharie	85	54
St Alhallowes Lesse	239	175	St Katherine Coleman	299	213
St Alhall Lumbardstr	90	62	St Katherine Cree-chu.	335	231
St Alhallowes Staining	185	112	St Lawrence Iewrie	94	48
St Alhallowes the Wal	500	356	St Lawrence Pountney	214	140
St Alphage	271	115	St Leonard Eastcheape	42	27
St And ew Hubbard	71	25	St Leonard Fosterlane	335	255
St Andrew Vndershaft	274	189	St Magnus Parish	103	60
St Andrew Wardrobe	476	308	St Margaret Lothbury	100	66
St Anne Aldersgate	282	197	St Margaret Moses	38	25
St Anne Blacke Friers	652	467	St Margar New Fishst	114	66
St Antholins Parish	58	33	St Margaret Pattons	49	24
St Austins Parish	43	20	St Mary Abch irch	99	54
St Bartl ol. Exchange	73	51	St Mary Ald rmanbury	181	109
St Bennet Fynch	47	23	St Mary Aldermary	105	75
St Bennet Grace chur	57	41	St Mary le Bow	64	36
St Bennet Pauls Wharf	355	172	St Mary Bothaw	55	30
St Benr et Sherehog	11	1	St Mary Colechurch	17	6
St Botolph B llingsgate	83	50	St Mary Hill	94	64
Christs Church	653	467	St Mary Mounthaw	56	37
St Christophers	60	47	St Mary Summerset	342	262
St Clements Eastcheaj	38	20	St Mary Sta nings	47	27
St Dionis Back-church	78	27	St Mary Wool hurch	65	33
St Dunstans East	265	150	St Mary Woolnoth	75	38
St Ed nunds Lumbard	70	36	St Martins Iremonger	21	11
St Ethelbo ough	195	106	St Martins Ludgate	196	128
St Fa ths	104	70	St Martins Orgars	110	71
St Fosters	144	105	St Martins Outwitch	60	34
St Gabri l Fen-church	69	39	St Martins Vintrey	417	349
St George Bot lphlar e	41	27	St Matthew Fridaystr	24	6
St Gregories by Pauls	376	232	St Maudlins Milkstreet	44	22
St H ll ns	108	75	St M udlins Oldfishstr	176	121

poisoning caused by one or other of the Salmonellae appear in every report and four were selected for special notice Severe smallpox occurred three times and mild smallpox once The other special reports concerned diphtheria and poliomyelitis It is a sign of the times that individual outbreaks of diphtheria can be singled out for special notice As recently as 1941 the disease was so common that it would have been impossible for the Chief Medical Officer to select individual outbreaks The special reports on poliomyelitis *relate to districts where prevalence was particularly high in a* restricted area in the Isle of Wight in 1950 and at Guildford in 1952

These then are the diseases which have been of particular concern to the Ministry of Health in recent years Poliomyelitis is the only one of them which has been epidemic in the country as a whole though outbreaks of food poisoning have been more common than they should be The other diseases may be described as rare specimens Fortunately all of them cause relatively few deaths and all except poliomyelitis can be prevented The production of a vaccine against poliomyelitis gives promise that in the next few years it may be possible to stop the spread of this disease

Buried in the 9 Parishes in the City and Liberties of Westminster 12194

Whereof of the Plague 8403

The Total of all the Christnings 9967

The Total of all the Burials this year 9 306

Whereof of the Plague 68,96

Diseases and Casualties this year

Abortive and Stilborne	617	Headmouldshot & Mouldfallen	14
Aged	1545	Jaundies	110
Ague and Feaver	5257	Impostume	227
Appoplex and Suddenly	116	Kild by several accidents	46
Bedrid	10	Kings Evill	86
Blasted	5	Leprosie	2
Bleeding	16	Lethargy	14
Bloudy Flux Scowring & Flux	185	Livergrowne	20
Burnt and Scalded	8	Meagrom and Headach	12
Calenture	3	Measles	7
Cancer Gangrene and Fistula	56	Murthered and Shot	9
Canker and Thrush	111	Overlaid and Starved	45
Childbed	625	Palsie	30
Chrisomes and Infants	1258	Plague	68,96
Cold and Cough	68	Plannet	6
Collick and Winde	134	Plurisie	15
Consumption and Tissick	4808	Poysoned	1
Convulsion and Mother	2036	Quinsie	35
Distracted	5	Rickets	557
Dropsie and Timpany	1478	Rising of the Lights	397
Drowned	50	Rupture	34
Executed	21	Scurvy	105
Flox and Smal Pox	655	Shingles and Swine pox	2
Found dead in streets fields &c.	20	Sores Ulcers broken and bruised Limbes	82
French Pox	86	Spleen	14
Frighted	23	Spotted Feaver and Purples	1929
Gout and Sciatica	27	Stopping of the Stomack	332
Grief	46	Stone and Strangury	98
Griping in the Guts	1288	Surfet	1251
Hangd & made away themselves	7	Teeth and Worms	2614
		Vomiting	51
		Wenn	1

	Buried	*Pla*		*Buried*	*Pla*
St Michael Bassishaw	253	164	St Olaves Iewry	54	32
St Michael Cornehill	104	52	St Olaves Siluerstreet	250	132
St Michael Crookedla	179	133	St Pancras Soperlane	30	15
St Michael Queeneh	203	122	St Peters Cheape	61	35
St Michael Querne	44	18	St Peters Corne hill	136	76
St Michael Royall	152	116	St Peters Pauls Wharfe	114	86
St Michael Woodstreet	122	62	St Peters Poore	79	47
St Mildred Breadstreet	59	26	St Stevens Colemanst	560	391
St Mildred Poultrey	68	46	St Stevens Walbrooke	34	17
St Nicholas Acons	46	28	St Swithins	93	56
St Nicholas Coleabby	1-5	91	St Thomas Apostle	163	110
St Nicholas Olaues	90	62	Trinitie Parish	115	[illegible]9
St Olaves Hart streete	237	160			

Buried in the 97 Parishes within the walls 15207
Whereof of the Plague 9887

	Buried	*Pla*		*Buried*	*Pla.*
St Andrew Holborne	3958	3103	St George Southwark	1613	1 60
St Bartholmew Great	493	344	St Giles Cripplegate	8069	4838
St Bartholmew Lesse	193	139	St Olaves Southwark	4793	2785
St Bridget	*2111*	*1427*	*St Saviours Southwark*	*4235*	*3446*
Bridewell Precinct	230	179	St Sepulchres Parish	4509	2746
St Botolph Aldersgate	997	755	St Thomas Southwark	475	371
St Botolph Algate	4926	4051	Trinity Minories	168	123
St Botolph Bishopsgate	3464	2500	At the Pesthouse	159	156
St Dunstans West	958	665			

Buried in the 16 Parishes without the Walls 41351

Whereof of the Plague 28888

	Buried	*Pla*		*Buried*	*Pla*
St Giles in the Fields	4457	3216	St Magdalens Bermon	1943	1363
Hackney Parish	232	132	St Mary Newington	1272	1004
St James Clarkenwel	1863	1377	St Mary Islington	696	592
St Katherines Tower	956	601	St Mary Whitechap	4766	3855
Lambeth Parish	798	537	Redriffe Parish	304	210
St Leonards Shorditch	[illegible]669	1949	Stepney Parish	8598	6583

Buried in the 11 out Parishes in Middlesex and Surrey 28554

Whereof of the Plague 21420

	Buried	*Pla*		*Buried*	*Pla*
St Clement Danes	1969	1319	St Mary Sauoy	303	198
St Paul Covent Garden	408	261	St Margaret Westmin	4710	3742
St Martin in the Field	4804	[illegible]883	*Thereof at the Pesthouse*	–	156

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APPENDIX

Christned	Males	5114
	Females	4853
	In all	9967
Buried	Males	48569
	Females	48737
	In all	97306
Of the Plague		68596

Increased in the Burials in the 130 Parishes and at the Pest house this year	79009
Increased of the Plague in the 130 Parishes and at the Pest house this year	68590

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